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ACUTE ILEUS *

ANALYSIS OF ONE HUNDRED THIRTY CASES OPERATED UPON AT THE PRESBYTERIAN HOSPITAL, NEW YORK CITY. FROM 1932 TO 1935, INCLUSIVE

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IN a recent report[1] on the mortality in cases of acute ileus the authors tabulated the results for 20 years (1916–1935, inclusive) by four-year periods and showed that there had been a reduction in general mortality from 66.6 per cent in the first four-year period (1916–1919, inclusive) to 27.6 per cent in the last four–year period (1932–1935, inclusive) at the Presbyterian Hospital in New York City.

The most striking reduction occurred in this last four-year period when the average mortality was found to be about 20 per cent less than in the preceding four-year period. A more complete analysis of this series of 130 cases seemed worthwhile in an attempt to account intelligently for the better results. A careful study was made of each case by both authors and the whole series was first divided into three groups, classified according to type: (1) Complete intestinal obstruction; (2) incomplete intestinal obstruction; and (3) paralytic ileus. The criteria for this classification were as follows:

(*a*) In the small bowel, obstruction was considered complete if the lumen was distended above and collapsed below the constriction.

(*b*) In the large bowel, obstruction was considered complete if the lumen was distended above and at the point of constriction was not more than

TABLE I

COMPARISON OF MORTALITY OF GROUPS CLASSIFIED ACCORDING TO TYPES IN A SERIES OF 130 ACUTE ILEUS CASES

	Total No.	Late Case Per Cent	Recovered	Died	Mortality Per Cent
Series	130	60%	94	36	27.6%
Complete obstruction	91	63%	78	13	14.3%
Incomplete obstruction	26	88%	15	11	42.3%
Paralytic ileus	13	69%	1	12	92.3%

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3 millimeters in diameter (as reported from the Surgical Pathology Laboratory or found at autopsy).

(*c*) The case was considered a paralytic ileus if the intestines were more or less uniformly distended, diffuse peritonitis was present and no obstruction was found. Recent adhesions angulated the bowel and caused complete or partial obstruction in 21 cases. These were classified as intestinal obstruction (mechanical ileus) whether or not active peritonitis was present, and are really a combination of mechanical and paralytic factors.

A comparison of the mortality in these groups is shown in Table I.

COMMENTS ON TABLE I

(1) This analysis and classification as to type of the ileus demonstrates strikingly how much more serious a condition paralytic is than mechanical ileus.

(2) It indicates that a patient's chances are better with a complete obstruction than with an incomplete obstruction.

(3) It indicates that the complete obstruction and the paralytic ileus cases are operated upon earlier than the incomplete obstructions when the symptoms appear less urgent and progressive.

(4) It shows conclusively that the group of complete obstructions is responsible for the total reduction of the mortality of the whole series. This, the largest of the three groups, has the smallest percentage of late cases and the lowest mortality.

Many other reports of similar series cases have shown striking variations in mortality of separate groups classified according to the exciting cause or mechanism of the ileus condition. The next step, therefore,

TABLE II

ANALYSIS OF GROUPS OF ACUTE ILEUS CASES, CLASSIFIED ACCORDING TO EXCITING CAUSE OF THE ILEUS

(I) *Acute Mechanical Ileus—Complete Obstruction*

	Recovered	Died	Mortality Per Cent
41 cases of old postoperative adhesions or bands (2 with gangrene of bowel)	40	1	2.5%
15 cases of recent postoperative adhesions (recent peritonitis)	13	2	13.3%
12 cases of carcinoma of colon	9	3	25.0%
17 cases of strangulated hernia (7 with gangrene)	11	6	35.3%
4 cases of volvulus of intestine	3	1	25.0%
1 case of intussusception	1	0	0.0%
1 case of gallstone	1	0	0.0%
91			

(II) *Acute Mechanical Ileus—Incomplete Obstruction*

6 cases of old postoperative adhesions or bands	4	2	33.3%
6 cases of recent postoperative adhesions (recent peritonitis)	4	2	33.3%
12 cases of neoplasm of intestine (10 carcinoma of colon)	5	7	58.3%
1 case of volvulus of intestine	0	1	100.0%
1 case of syphilis of intestine	1	0	0.0%
26			

(III) *Paralytic Ileus—No Obstruction Found*

5 cases of acute diffuse peritonitis following acute appendicitis	1	4	92.3%
3 cases of acute diffuse peritonitis following resection of carcinoma	0	3	
1 case of acute diffuse peritonitis following acute pancreatitis	0	1	
1 case of acute diffuse peritonitis following acute diverticulitis	0	1	
1 case of acute diffuse peritonitis following strangulated hernia	0	1	
1 case of acute diffuse peritonitis following acute salpingitis	0	1	
1 case of acute diffuse peritonitis following mesenteric thrombosis	0	1	
13			

was to divide our three primary groups of complete obstruction, incomplete obstruction and paralytic ileus into subgroups classified on a basis of causal mechanism. Table II shows the mortality percentage in these subgroups.

COMMENTS ON TABLE II

(1) It will be seen that nearly one-half of the complete obstruction group were caused by old peritoneal bands or adhesions, requiring relatively brief and simple operations. On the other hand, nearly one-half the cases in the incomplete obstruction group were due to neoplasms requiring more prolonged and difficult operative procedures.

(2) It is at once evident that the large subgroup of cases due to old postoperative peritoneal adhesions or bands with their very low average mortality is responsible for the low general mortality of the primary group of complete intestinal obstruction.

(3) It is equally evident that the large proportion of intestinal neoplasms, with their high death rate, is largely responsible for the high general mortality of the primary group of incomplete intestinal obstruction.

(4) It is more difficult to explain why the 12 neoplasms causing complete obstruction had a mortality only one-half that of the neoplasms causing incomplete obstruction. It is also difficult to see why old adhesions causing complete obstruction had so small a mortality compared with those causing incomplete obstruction. It may have been due to individual variations or perhaps to the already noted fact that the incomplete obstructions were in general operated upon later than the complete obstructions.

(5) Cases due to recent postoperative adhesions (frequently complicated by a persisting peritonitis) had a much higher mortality when the obstruction was incomplete. It is not convincing but it suggests that peritonitis is an exceedingly serious complication of acute ileus.

(6) This suggestion is markedly emphasized by a glance at the paralytic ileus group, with one single recovery out of 13 cases when associated with acute, diffuse peritonitis following various causes.

(7) Because one finds similar variations in mortality dependent upon the cause or exciting mechanism of the ileus in reports by other writers, it would appear that, in order to compare fairly the results in one series of ileus cases with another, each series must first be analyzed in three ways:

(*a*) According to the percentage of late cases.

(*b*) According to the cause or mechanism.

(*c*) According to type; complete or incomplete mechanical or paralytic ileus. If this is not done, three criteria for intelligent judgment have been omitted.

TABLE III

ANALYSIS OF ADJUVANT TREATMENT IN 91 CASES OF ACUTE ILEUS OPERATED UPON AT THE PRESBYTERIAN HOSPITAL, 1932–1935, INCLUSIVE

Diagnostic Roentgen Ray	Type of Anesthesia			Gastric Lavage or Nasal Tube	Replacement of Fluids and Electrolytes	Use of Peristaltic Drugs	Rectal Treatments
Taken on all patients where diagnosis was not obvious. Roentgenograms were positive in 92%, negative or doubtful in 8% of cases in which obstruction was proved at operation	Local	General	Spinal	Used only in 40 cases	All but 3 patients had 2,200 cc. average given q.d. for an average time of 5 days postop. Fluid used was generally normal saline, occasionally hypertonic saline, very rarely 5% glucose	60 patients had peristaltic drugs postop. An average of 4 to 5 doses a day for an average time of 5 days	67 patients had colon lavage
	15 cases	36 cases	40 cases	Mort. 25%		Mort. 11.7%	Mort. 15%
	Mort. 27%	Mort. 33%	Mort. 10%			31 patients were given none	24 patients had none
						Mort. 33.3%	Mort. 37.5%

The results were so much better in the group of cases of complete obstruction in the series we are reporting here, that we made an analysis of the adjuvant treatment in these 91 cases, seeking to discover further light on the cause of the improvement in mortality. This analysis is shown in Table III.

COMMENTS ON TABLE III

(1) *Diagnostic Roentgenograms* in three positions (supine, and lying on right and left sides), were taken in 78 cases where the diagnosis was not obvious. In 92 per cent of these cases they made the diagnosis positive at once, and obviated the delays of further investigation, thus predisposing to earlier operation and better results.

(2) *Anesthesia.* It would appear from this table that local anesthesia, with a mortality of 27 per cent in the cases where it was used, was dangerous. The appearance is deceptive, however. Local anesthesia was used in a few cases of strangulated hernia but it was employed mostly for the preliminary cecostomy in obstruction due to carcinoma or for secondary enterostomy in cases where the relief operation had not been completely successful. Such cases have what might be called a natural high mortality and it would be unfair to ascribe it to the local anesthesia.

General Anesthesia was used in 36 of the operations with a mortality of 33 per cent; avertin supplemented by nitrous oxide and oxygen 15 times; nitrous oxide and oxygen supplemented by ether 14 times; avertin supplemented by local anesthesia three times; and nitrous oxide and oxygen supplemented by local four times. It was used in most of the carcinoma cases where a later resection followed the preliminary cecostomy.

Spinal Anesthesia (generally novocain [100–200 mg.] or pantocain 2 cc.) was used altogether 40 times; approximately in the same number of herniae, adhesion-obstructions and peritonitis cases as was general anesthesia. It was used in only half as many cases of carcinoma. The mortality of ten per cent contrasts favorably with that of the general anesthesia, particularly as they were used in similar types of cases.

Certainly there is a real advantage in having a patient conscious, if he is liable to vomit. Theoretically, there is also an advantage in having the peristaltic stimulation of the bowel which often follows spinal anesthesia.

(3) *The Nasal Stomach Tube* for suction and irrigation was used comparatively little, until the year 1935. It was reported as being effective and helpful but was used in so small a percentage of the patients that no more definite report can be made. It is now being used much more frequently.

(4) *Replacement of Fluid and Electrolytes.* This procedure has been followed for a number of years but not so frequently and persistently as it has been for the past four or five years. The smallest amount given any one patient was 1,000 cc. of saline in one day. The largest total amount given to any one patient was 77,000 cc. in 30 days. Administration was by hypodermoclysis, by repeated infusions or by continuous infusion, supplemented by proctoclysis.

(5) *Peristaltic Drugs.* These have been used much more frequently during the past four or five years as a postoperative routine, except in cases where lower bowel resection had been done. Two-thirds of the patients were given them. They were combined with rectal treatment, usually colon lavage, less frequently enemata or irrigations. The smallest amount given to any patient was six doses of pitressin, in one day. The largest amount was 64 doses, in 13 days. Pitressin was most frequently used, sometimes prostigmine, occasionally pituitrin and rarely peristaltin.

(6) *Rectal Treatment.* Sixty-seven patients received postoperative rectal treatment. Twenty-four patients did not. The latter group comprised those who had had colon resection and those whose peristalsis reëstablished itself automatically.

It is unwise to be too definite in commenting on these factors of treatment but one

feels justified in saying that the following *appear* to have had a favorable effect on the mortality:

(1) The routine use of diagnostic roentgenologic examination.
(2) More frequent use of spinal anesthesia.
(3) The more generous replacement of fluid and NaCl.
(4) The more frequent administration of peristaltic drugs after operation.

We believe that there is another important factor making for improvement; namely, the greater general interest in, and the better understanding of, these cases which have resulted from their more intensive study.

OPERATIVE TREATMENT

The operative treatment in these 91 cases varied from the cutting of a single obstructing band to an extensive resection of the colon for carcinoma, and was too varied to classify, usefully, with the exception of the operation of enterostomy, which was performed 42 times in 39 cases.

Table IV exhibits the analysis as to time (primary, done at the time of relief operation, or preliminary to it; and secondary, done after relief operation, to supplement it), as to method (Witzel or Kader), and as to location (jejunum, ileum, colon). The mortality percentages stated are not of great value because the number of cases is so small.

TABLE IV

ANALYSIS OF 42 CASES OF ENTEROSTOMY PERFORMED UPON 39 PATIENTS WITH COMPLETE OBSTRUCTION AT THE PRESBYTERIAN HOSPITAL, 1932–1935, INCLUSIVE

Time		Method		Location		
Primary	Secondary	Witzel	Kader	Jejunum	Ileum	Colon
30 cases	12 cases	21 cases	21 cases	21 cases	6 cases	15 cases
23.3% mortality	23.0% mortality	15% mortality	23.8% mortality	23.8% mortality	33.3% mortality	20% mortality

COMMENTS ON TABLE IV

(1) These 39 patients upon whom enterostomy or colostomy was performed were undoubtedly the more seriously sick among the 91 cases analyzed as to treatment. They included the great majority of the carcinoma and peritonitis cases, which accounts in part for their high average mortality.

(2) Their average mortality was 28.2 per cent as contrasted with an average mortality of 3.8 per cent among the 52 cases, without enterostomy, in the same group of complete obstructions.

(3) All but one of the enterostomies were reported as being effective (from the point of view of discharging bowel contents and relieving intra-intestinal pressure) and only two had to be closed by operation (the gunbarrel type). But there were three cases in which two enterostomies (each) were performed. Out of the total 42 operations performed, it would be safe, therefore, to assume that 39 were effective and three unsatisfactory.

(4) Because of the small number of cases in each column of this table, and because of modifying conditions, we do not feel justified in drawing any definite conclusions

as to the effect on the mortality percentage of the method used, or the location of the opening.

(5) We do, however, believe that the time is important and that a primary enterostomy or colostomy, performed as a part of the release operation (or preliminary to it), is more effectual than a secondary one, performed later to supplement an unsatisfactory release operation. And we further believe that in cases of obstruction above the ileocecal junction an opening in the jejunum is better than in the ileum because the upper bowel, being farther away, is less affected by the obstruction and more likely to function effectively in ridding itself of overdistention through the opening.

(6) Enterostomy should be thought of, and employed, only as a safety valve to relieve or to prevent overdistention of the bowel by gas or fluid when that is present or expected. And it must be remembered that, just as with a safety valve set too low, it may occasionally result in loss of vitally useful contents. It has not been employed with sufficient judgment and discretion. We still believe that in certain selected cases it is to be recommended provided it is done early enough.

(7) The relative ineffectiveness of enterostomy in paralytic ileus and in late cases of obstruction is seen in Table V, where the mortality of enterostomy in cases of complete obstruction is compared with that in cases of incomplete obstruction (most of which were late) and in cases of paralytic ileus.

TABLE V

COMPARISON OF RESULTS OF ENTEROSTOMY IN I, COMPLETE OBSTRUCTION; II, INCOMPLETE OBSTRUCTION; III, PARALYTIC ILEUS

COMPLETE OBSTRUCTION

I. 42 enterostomies (performed on 39 out of 91 cases of complete obstruction) ..... Mortality 28.2%

INCOMPLETE OBSTRUCTION

II. 17 enterostomies (performed on 17 out of 26 cases of incomplete obstruction) ..... Mortality 53.0%

PARALYTIC ILEUS

III. 10 enterostomies (performed on 10 out of 13 cases of paralytic ileus). Mortality 90.0%

CAUSES OF DEATH

The list of deaths shown in Table VI emphasizes the well recognized fact that comparatively few cases of mechanical ileus die from the obstruction itself but rather from the complications of the ileus. After the obstruction has been relieved most of the deaths are attributable to pneumonia or peritonitis.

TABLE VI

ANALYSIS OF CAUSE OF DEATH AND LENGTH OF POSTOPERATIVE SURVIVAL IN 130 CASES OF ACUTE ILEUS OPERATED UPON AT THE PRESBYTERIAN HOSPITAL, 1932–1935, INCLUSIVE

COMPLETE INTESTINAL OBSTRUCTION

Unit Hist.	
458538	Strangulated femoral hernia (C. Welchii, hemolytic colon B. and *Streptococcus viridans* in sac with gangrenous intestine), (herniotomy). 63-year-old woman. Died of peritonitis 36 hours postoperative.
423898	Strangulated inguinal hernia, (herniotomy). Died pneumonia, 12 days postoperative.

TABLE VI (*Continued*)

COMPLETE INTESTINAL OBSTRUCTION (*continued*)

Unit Hist.	
81621	Strangulated umbilical hernia, (herniotomy). Died six hours postoperative, probably of cardiac failure (very fat man 5 ft., 4 in., weighing 229 lbs.).
350920	Strangulated femoral hernia, (herniotomy). Died nine days postoperative, probably of pneumonia.
373120	Carcinoma of sigmoid (spontaneous perforation, acute diffuse peritonitis), (exploratory celiotomy). Died six days postcecostomy of peritonitis.
384828	Pelvic abscess, (drainage). Died 37 days postoperative, probably asthenia (75 years old).
394664	Acute appendicitis; intestinal obstruction by adhesions, (release of adhesions, drainage of abscess). Died of suppurative pyelophlebitis 30 days postoperative.
398018	Strangulated inguinal hernia; gangrenous intestine, (gunbarrel resection). Died 18 hours postoperative, probably of peritonitis.
400609	Meckel's diverticulitis; foreign body ileus, (resection). Died peritonitis and pneumonia 11 days postoperative.
389690	Carcinoma of cervix, (radium, ileocolostomy). Died 67 days postoperative of carcinoma and radium necrosis.
315483	Carcinoma of stomach, (resection, adhesions, volvulus). Died three days postoperative of peritonitis.
335827	Carcinoma of sigmoid, (cecostomy, colostomy). Died of pneumonia, 16 days postoperative. (old cardiac, 69 years old.)
333373	Strangulated femoral hernia; with gangrene of ileum, (partial enterectomy, ileostomy). Died 17 days postoperative of pneumonia (85 years old).

INCOMPLETE INTESTINAL OBSTRUCTION

369406	Carcinoma cecum, (4 hour resection). Died five days postoperative of peritonitis.
421285	Carcinoma stomach, (resection, peritoneal abscess). Obstruction 15 days postoperative. Died of carcinoma and peritonitis 27 days postoperative.
458227	Carcinoma of colon, (no resection). Died 13 days postcecostomy of uremia and lobular pneumonia.
347720	Carcinoma sigmoid, recurrent, (resection). Died 21 days postoperative of pneumonia.
379322	Acute appendicitis. Acute diffuse peritonitis, (jejunostomy, drain abscess, appendicectomy). Died 16 days postoperative of pneumonia.
330777	Carcinoma of sigmoid, (cecostomy). Died three days postoperative of pneumonia (73 years old).
369480	Reticulum cell sarcoma of colon and ileum, (exploratory celiotomy, ileostomy and colostomy). Died 14 days postoperative of sarcoma of peritoneum of bowels. (Age 66.)
258705	Ulcer of stomach, (Polya resection of stomach). Postoperative kinking of jejunum and leakage of suture line. Died of peritonitis and ileus 12 days postoperative.

TABLE VI (*Continued*)

INCOMPLETE INTESTINAL OBSTRUCTION (*continued*)

Unit Hist.	
349836	Ventral hernia and postoperative adhesions, (repair of hernia and division of adhesions jejunostomy). Died 15 days postoperative of peritonitis of unknown origin.
335621	Carcinoma of transverse colon and rectum, (resection of transverse colon. Abdominal perineal proctectomy eight days later). Died three days after second operation.
333488	Carcinoma of sigmoid with metastases and pul. tbc., (cecostomy, palliative). Died 16 days postoperative of pneumonia.

PARALYTIC ILEUS

386886	Strangulated femoral hernia, (herniotomy wound disruption, peritonitis) Died 19 days postoperative of peritonitis.
339700	Mesenteric thrombosis, gangrenous ileum, acute diffuse peritonitis, (resection). Died, extension of thrombosis, two days postoperative.
384874	Carcinoma of rectum, (abdominal perineal resection). Died 19 days post-resection, probably of peritonitis and paralytic ileus.
341572	Pelvic abscess with acute diffuse peritonitis, (drainage of abscess, ileostomy). Died of peritonitis (following perforation of ileum by enterostomy tube) four days postoperative.
419365	Acute appendicitis, disruption of operative wound, (appendicectomy, repair of disrupted wound, jejunostomy). Died six days postoperative of pneumonia and peritonitis.
227507	Acute pancreatitis, acute diffuse peritonitis, (cholecystectomy, chronic cholecystitis with calculi, choledochotomy). Died nine days postoperative of peritonitis.
339864	Carcinoma of rectum, (general arteriosclerosis), (colostomy, enterostomy). Died of paralytic ileus four and one-half days postoperative. (Age 71.)
347615	Acute appendicitis, acute diverticulitis with abscess, acute diffuse peritonitis (drainage of abscess, cecostomy, jejunostomy). Died 15 days postoperative of peritonitis.
433333	Carcinoma of cecum with metastases, (resection of cecum, jejunostomy, revision of jejunostomy). Died four days postoperative of shock and peritonitis.
350959	Acute salpingitis, acute diffuse peritonitis, (appendicectomy, salpingo-oöphorectomy). Died 24 hours postoperative of peritonitis and shock.
402686	Diverticulitis of sigmoid, acute diffuse peritonitis, (exploratory celiotomy, ileostomy). Died six days postoperative of peritonitis.
336060	Acute appendicitis, acute diffuse peritonitis, (celiotomy with drainage). Died of pneumonia and uremia five days postoperative.

CONCLUSIONS

(1) The careful study and analysis of a series of acute ileus cases has been helpful in reducing their mortality percentage.

(2) The factors chiefly responsible for the improvement seem to be:

(*a*) Earlier operation; partly due to routine preoperative roentgenologic examination.

(*b*) Better understanding of the problem to be solved.

(*c*) More frequent use of spinal anesthesia.

(*d*) Greater replacement of fluids and electrolytes.

(*e*) More frequent use of peristaltic drugs and rectal treatments.

(3) Continued improvement will probably depend largely upon earlier diagnosis.

REFERENCE

[1] vanBeuren, F. T., and Smith, B. C.: Mortality in Acute Ileus, ANNALS OF SURGERY, 106:752–755. October, 1937.

RÉSUMÉ BY DR. BEVERLY C. SMITH

(1) Mortality 66.6 per cent, 1916–1919 (acute ileus cases at Presbyterian Hospital) and 27.6 per cent, 1932–1935; 14.3 per cent in the acute mechanically obstructed group.

(2) The advisability of two persons studying and classifying the charts of the cases included, to determine as closely as possible, from all available sources in the chart: (a) The duration of the obstruction; (b) the type, *i.e.*, mechanical or paralytic—the degree of each if coexistent—and when the paralytic type became superimposed upon the mechanical; and (c) the degree of mechanical, *i.e.*, whether complete or incomplete.

The charts, about which either Doctor vanBeuren or Doctor Smith was in doubt, were reviewed individually and together before the case was finally classified. Record Room and discharge diagnoses were so frequently inadequate or incorrect that they could not be relied upon for statistical data. The criteria for complete and incomplete mechanical and paralytic ileus as outlined in the paper were accepted because of their simplicity and recurrent accuracy.

(3) 14.3 per cent mortality in 91 cases of complete obstruction is a surprising and unusual percentage.

(4) It is important to contrast this with a mortality of 42.3 per cent resulting in 26 cases of incomplete obstruction. The difficulty of diagnosing the incomplete, as compared to the complete variety is emphasized, with the delay in operating upon the incomplete cases because of uncertain diagnosis acting as the greatest contributory cause of the high mortality in this group.

(5) The high incidence of postoperative adhesions—41 or 45 per cent of 91 cases of complete obstruction—strikingly illustrates the necessity of thoughtful peritonealization, gentle padding and handling of viscera, respect for the omentum, and serious consideration of the type and duration of drainage in each case.

(6) In the incomplete group of 26 cases the highest mortality occurred in 12 cases of neoplasm of the intestine of which 10 were carcinomas of the colon. The mortality of 58.3 per cent was caused by the high incidence of peritonitis following resection.

(7) The paralytic cases were all associated with peritonitis and the mortality of 92.3 per cent is a striking warning as to the anticipation and

early treatment directed towards the prevention of this most serious complication.

(8) The most treacherous and difficult type of case to diagnose was that due to recent postoperative adhesions associated with a peritonitis. The possibility of obstruction in such cases should be borne in mind and every effort should be made to note its presence as early as possible.

(9) We suggest for the consideration of future essayists on this subject that they consider similar criteria of classification, namely:

(a) The percentage of late cases, *i.e.,* over 48 hours' duration,
(b) The cause or mechanism of the obstruction,
(c) The type—complete, incomplete, mechanical or paralytic.

(10) As to diagnostic methods:

(a) A careful history remains preëminent.
(b) The roentgenologic examination has been most important and very reliable (92 per cent positive in 78 cases). It was correct in 96 per cent cases in which it was employed and which were operated upon. It was not used in the more obvious type of cases such as strangulated herniae.

The roentgenogram may be equivocable or negative—it is not infallibly correct, but when negative the operative indications again must revert to the history, physical signs and response to treatment.

(11) Anesthesia.—Spinal anesthesia seems to have proven most satisfactory in this group. Yet there are definite contraindications to the use of it in certain types of cases and individuals. Two cubic centimeters of pantocain in the second or third lumbar spaces was most frequently used. A combination of spinal and some type of inhalation anesthesia gave the greatest percentage of postoperative respiratory and wound healing complications.

(12) The Nasal Tube:

(a) A few patients will not tolerate it.
(b) Some patients vomit the tube repeatedly.
(c) Ulceration in the esophagus has been noted at postmortem in some of the cases in which the tube was used.
(d) One case with a deviated septum had an ulceration of the mucous membrane on the septum and an adhesion to the lateral nasal wall formed which nose and throat consultants thought could not be cured without a septal resection.
(e) Cases of alkalosis from prolonged use of the tube have been reported—at the proper time it may be clamped off for periods.
(f) It may relieve symptoms preoperatively temporarily, while a loop of gut with damaged blood supply remains in the abdomen.

Preoperative gastric lavage is particularly indicated to deflate the stomach and prevent aspiration of vomitus during induction and recovery from anesthesia.

Continuous preoperative nasal tube suction was most beneficial but it will not always empty the lower small intestine and if used for a prolonged period the plasma chlorids and CO_2 must be watched for possible alkalosis. A 2 per cent cocaine spray to the nose and nasopharynx facilitates the passage of the tube with the minimum of gagging and strain on the abdominal wall wound. A tube may be curled in the stomach and its open end be above the most dependent portion of the stomach. Its position should be checked by a roentgenogram or the fluoroscope if it does not function. The time to withdraw the tube can largely be determined by the character of the drainage material. Its replacement should be constantly borne in mind.

(13) Replacements of fluids and electrolytes. The size and weight of the patient, the functional capacity of the myocardium and kidneys, the degree of shock, and the insensible fluid loss should be carefully estimated, and the plasma chlorides and serum protein and blood specific gravity should be followed to maintain a patient in water, electrolyte and protein equilibrium. An average total parenteral intake of four to six liters of normal saline solution for a few days seems indicated in the average adult case.

(14) The postoperative hypodermic administration of peristaltic drugs, such as pitressin, prostigmine, pituitrin or peristaltine combined with the judicious employment of colon irrigations, lavages and enemata have, clinically, improved peristalsis, and their use should be persisted in until deflation and successful responses are consistently obtained.

(15) The nasal tube seems to have replaced enterostomy in many of our cases. Whereas this paper does not contain statistical data on enterostomies, we did note, in this series, that a primary enterostomy was more effective than a secondary one, and an ostomy in the more viable jejunum seems to function better than one in the more involved ileum. The clinical and pathologic indications for enterostomy require astute judgment. The relative ineffectiveness of enterostomy in paralytic ileus is stressed: (a) The mortality following 42 enterostomies performed upon 39 cases out of 91 acute mechanical obstructions was 28.2 per cent. (b) In 17 enterostomies performed upon the group of 26 cases of incomplete obstruction the mortality was 53 per cent. (c) In ten enterostomies performed upon the group of 13 cases of paralytic ileus the mortality was 90 per cent.

CASE REPORTS ILLUSTRATING ACUTE ILEUS

Presented by Dr. Beverly C. Smith

Case 1.—W. R., age 45 (1923), now 59 (U. H. No. 57572), was admitted to Presbyterian Hospital July 23, 1923, complaining of having had attacks of cramping abdominal pains for the previous three years. In 1917, he had been operated upon at the Polyclinic Hospital for a gastric ulcer. Supposedly a gastro-enterostomy and an appendicectomy were performed. He was well for three years thereafter, until 1920, when the present illness began, which has been characterized by sudden, severe attacks of upper abdominal cramp-like pain with vomiting. He was admitted to, and remained in Mount Sinai Hospital for 11 days during this attack, which subsided under symptomatic treatment.

Seven months later a similar episode occurred which gradually disappeared without treatment. He vomited food eaten, then green fluid during this attack.

The free interval between attacks had become shorter, but their severity did not increase until, on July 3, 1923, his pain became so severe that he fell, unconscious, in the street, and was taken to the Jamaica Hospital, and was discharged the following day. He had another attack July 20, 1923, to control which morphine was administered. This was followed three days later by a similar seizure which subsided spontaneously.

Because the patient thought that food caused his attacks, he had not eaten for two days before admission to the Presbyterian Hospital, July 23, 1923. He had been on a modified diet without alkaline medications ever since his operation for gastric ulcer, although six months previously his ulcer had been pronounced healed which was confirmed by a gastro-intestinal roentgenologic series. He had lost 28 pounds during the two months prior to admission, due to starvation.

His family history was negative, and his past history revealed that he had suffered from indigestion since age 25.

Physical Examination.—Blood pressure 108/70. Weight loss evident. The teeth were carious and pyorrhea was present. Abdominal examination was negative except for a right rectus scar, without herniation. Rectal examination was negative. *Provisional Diagnosis.*—Chronic ileus.

Laboratory Data—Blood urea, July 25, 1933: 0.34 Gm./L. Wassermann: Negative.

	Free Hcl.	Total Acid
Ewald test meal:		
Fasting expression.....	0	18
Expression 1 hr. after meal.....	0	30
Expression 2½ hrs. after meal.....	0	36

Red blood cells, 4,500,000; hemoglobin, 100 per cent; white blood cells, 6,800; polymorphonuclears, 54 per cent.

Gastro-intestinal series, July 31, 1923, revealed a 15 per cent, six hour, gastric residue. A gastro-enterostomy stoma was present, the barium passing both through it and through the pylorus. In six hours the barium had reached the terminal ileum. Two loops of ileum appeared dilated but the barium was not delayed in them. In 24 hours, the ascending, transverse, descending colon and rectum were normally filled.

After five days of hospitalization the patient was discharged to the Surgical Diagnostic Clinic for a further work-up. Letters received from various doctors and hospitals confirmed the patient's story and diagnosis. No mention of obstruction was made in any of them.

At a three and six months follow-up, he had been continuously asymptomatic. At 12 months, attacks had recurred. Three followed in the next four months, characterized by sudden cramping pain just below the umbilicus and the vomiting of green fluid. His physician, during each attack, gave him a hypodermic of morphine which allowed him to sleep and when he awakened the pain had gone.

He was readmitted to Presbyterian Hospital, on August 28, 1925, complaining of a sudden, excruciating attack of crampy pain in his upper abdomen which had begun 16 hours previously. Soon after its onset he vomited material eaten six hours before and had vomited greenish fluid repeatedly since. The intensity of the crampy pains had diminished but they were still present intermittently. He had had a normal stool the morning of admission which had no effect on his pain. He was weak and prostrated. For four days he had had an acute cold with a productive cough.

Physical Examination showed a somewhat prostrated man with a pale, cold skin, bluish hands and dusky gray nail beds. He vomited yellowish-brown fluid without fecaloid odor twice while being examined, and cried out and writhed when a spasm of pain would come on. The lungs were normal. Blood pressure, 146/20. The abdomen was scaphoid rather than distended. There was no tenderness, spasticity or palpable mass. He had had a stool as the result of an enema and had passed a considerable amount of

flatus with a colon lavage. While being examined he had an attack of pain which made him cry out, and his upper right rectus became rigid, but as soon as the spasm subsided his abdomen immediately became soft again with no residual tenderness. It was this attack of pain, and his history—not his physical signs—which made us urge the advisability of an immediate celiotomy.

The operative findings were a surprise as compared to his preoperative signs and vividly emphasized the deceit and treachery of intestinal obstruction. No scout roentgenograms were made. Their proper interpretation would certainly have assisted greatly in the diagnosis.

Operation.—Under gas-oxygen anesthesia the abdomen was opened through an upper midline incision to the left of the right rectus scar. The jejunum with a cherry-red-bluish color, distended to four or five centimeters, and filled the right abdomen and pelvis. Seventy-five cubic centimeters of clear yellow peritoneal fluid was aspirated from the pelvis. A segment of midjejunum was adherent to the site of the old scar for a distance of four centimeters. This had acted as a fulcrum point about which the proximal jejunum had volvulated and descended into the pelvis. The distension was due mostly to fluid in the bowel and not to gas. The fluid made the loops so heavy that as one was drawn out of the cavity the weight of the fluid in it pulled more gut out, tending to eviscerate the patient. An enterostomy was done and a fenestrated No. 16 F catheter was introduced near the end of the volvulated jejunum in nearly normal appearing gut. The end of the catheter was brought out through a small stab wound. This did not drain the gut. A second enterostomy, therefore, was made higher up in the distended jejunum and 500 cc. of foul, brown fluid drained immediately. This permitted further exploration without evisceration, which revealed a collapsed stomach, colon and ileum, and no evidence of peritonitis or other adhesions. The adherent knuckle of gut was freed and peritonealized. The wound was closed in layers with chromic catgut. An infusion of a liter of normal saline and 500 cc. of 5 per cent glucose was introduced during the operation. Two rubber dam drains were placed in the subcutaneous wound because of possible contamination from an enterostomy tube.

Convalescence was relatively uneventful except for a wound infection which necessitated opening the skin incision, and resulted in a slough of some of the midline aponeurosis; evisceration did not occur, however, notwithstanding an exacerbation of his cough and bronchitis.

The lower jejunostomy tube was removed on the eighth day. No drainage followed and it remained healed. The upper jejunostomy tube was removed on the twelfth day and it too healed without subsequent drainage.

The appended tabulation shows an interesting comparison of the amount of drainage recovered from these tubes.

	1st 6 hrs.	1st day	2nd	3rd	4th	5th	6th	7th	8th	9th	10th
Upper tube....	605 cc.	110	170	60	10	20	120	125	250	350	200
Lower tube....	50 cc.	260	380	1,040	1,560	430	410	200	60		

Postoperative treatment consisted of 2,500 to 4,000 cc. of normal saline daily, continuous abdominal poultices, colon irrigations for two days, then enemata. Peristaltin was given twice on the third and fourth days, and rectal treatments were effective. On the twelfth postoperative day the patient vomited 800 cc. of yellow fluid, and on the fourteenth, eighteenth and twenty-first postoperative days 100 cc. of similar material. During this time rectal treatments were effectual and he did not become distended. He was discharged 29 days after operation.

Follow-up at two months: Rating 444.*

Follow-up at one year: He had had four attacks of abdominal cramps with vomiting.

* Anatomic 4 = 100%; symptomatic 4 = 100%; economic 4 = 100%.

Passing flatus and vomiting had relieved his pain. He had lost 19 pounds in weight and did not eat because he felt better when his stomach was empty.

In March, 1926, he was admitted the third time complaining of attacks of crampy pain and vomiting for the past two weeks. Pain began in the right flank and radiated into the epigastrium. Previous to this he had been symptom-free. The vomiting consisted of food eaten, followed by green fluid. It relieved his cramps and some days he vomited as frequently as seven times. His bowels had moved without catharsis up to the last week. Since then daily enemata have been necessary.

Physical Examination shows loss of weight. Foul breath odor. Abdomen flat, no distention. Two operative scars in upper abdomen. No tenderness, mass, visible peristalsis, or palpable viscera.

Laboratory Data: Blood pressure, 120/80; white blood cells, 11,400; polymorphonuclears, 63 per cent. Gastric lavage, clear-odorless green fluid. Blood urea, 14.2 mg. Plasma chlorides, 5.45 Gm. CO_2 51 Ml. Urine, negative.

Ewald meal:
Fasting. No free Hcl.
1 hr. after meal. free 11 ..... Total 45
2 hrs. after meal. free 0 ..... Total 30

He remained in the hospital six days. His signs and symptoms disappeared with gastric lavages and small fluid feedings.

Follow-Up.—One year after operation: Gained 14 pounds. Doing full days' work as a truck driver. Rating 444. Two years after operation: Has had three attacks of abdominal pain. Vomited green fluid several times with each attack. These attacks caused him to lose jobs as each incapacitated him for two to three days. Three years after operation: He has had one severe, similar attack which lasted 36 hours. He vomited six times with it. Cramps gradually subsided after a stool. Four years after operation: No attack for six months. Rating 444. He has developed a postoperative, medial, ventral wound hernia resembling a diastasis. It is symptomless and not enlarging. Five years after operation: Rating 444.

Eleven and one-half years after operation: Has been perfectly well until two months ago when he noted dizziness, weakness, nausea and vomiting. He has lost 18 pounds in past year. When he has abdominal pain he can occasionally feel a lump in the left upper abdomen.

Fasting gastric expression showed no free HCl. After an Ewald meal the free HCl was six and the total acid 16. The fasting expression of 100 cc. was a foul smelling, yellow, mucoid fluid.

October 9, 1937.—Fluoroscopy of his stomach showed a functioning gastro-enterostomy stoma and pylorus, without evidence of ulcer at either site. Barium passed through the small intestine without obstruction and reached the cecum in nine hours, slightly, but not appreciably delayed. The colon was normal. There was no roentgenologic evidence at this time of partial obstruction. Segmented distribution of the barium in his gastrointestinal series possibly indicates some degree of vitamin deficiency.

COMMENT.—This patient is presented as a case of recurrent, partial intestinal obstruction due to postoperative adherence of the gut to the wound site. It illustrates the value of the history in these cases and the difficulty in correlating the pathology in the gut with the physical signs. This patient had been seen by a number of doctors and had been in several hospitals. He had refused operation because he had recovered so rapidly from his attacks. It seems advisable to keep the cases under frequent observation during his attacks, and with the employment of roentgenologic examinations it may be easier to determine whether the pains are due to peristalsis and obstructed

bowel, or to peristalsis associated with peritoneal adhesions without obstruction. As will be seen in the statistical résumé, this type of case gives a higher percentage of mortality.

Case 2.—(U. H. No. 520466): B. G., female, age 64, was admitted to Presbyterian Hospital June 4, 1937, complaining of vomiting, upper abdominal pain, and obstipation of four days' duration. She had been operated upon by Dr. John B. Murphy at the Haneman Hospital in Chicago 40 years previously for the removal of a pelvic tumor, the nature of which she did not know. She had had typhoid fever in 1930, from which she recovered without complications. One week before admission she had an acute onset of pain in her right upper abdomen and flank which was followed by a chill. A saline cathartic and a hot water bottle applied locally gave her relief, and the next day she was free of pain.

Five days before admission she noted abdominal fullness associated with eructations, gas and obstipation, followed two days later by nausea and vomiting of first, mucoid, then green fluid. The vomiting usually occurred in the evenings and she was able to take and retain some food in the morning. She had had two stools in the five days before admission and had not taken any cathartic. Cramping pains originating in her right abdomen and radiating to her right back and shoulder had persisted for five days before admission.

At no time did this patient consider herself sufficiently ill to call a doctor. For years she had noticed an intolerance to fatty foods, constipation and bloating. During the past 15 weeks she had lost 15 lbs., primarily because of dieting in order to relieve her gastric distress.

Six weeks previous to admission her physician had diagnosed her case as one of hypertensive disease because of dyspnea on exertion and had given her digitalis. She had no history of cardiac decompensation. Her other systems were negative except that she had had a short period of unconsciousness nine weeks previous to admission, without paralysis or other sequelae. On admission she was slightly dyspneic but in no apparent acute distress.

Physical Examination.—Enlarged heart with extra systoles: apical systolic murmur with loud second aortic sound. Abdomen slightly distended with moderate tenderness in the right upper quadrant, most marked below the costal margin over the gallbladder region. No masses could be felt. A lower abdominal, midline scar was firm without evidence of hernia. Rectal and pelvic examinations were negative. A diagnosis of an acute exacerbation of a chronic cholecystitis had been made on admission.

Roentgenologic examination, June 4, 1937 (No. 159879), of the abdomen in three positions did not show dilated loops or fluid levels.

Laboratory Data.—Blood sugar and urea nitrogen, June 5, 1937, normal. EKG, June 5, 1937: Slight evidence of myocardial damage. Wassermann, negative.

June 7, 1937: Cholecystogram showed a faint gallbladder shadow without evidence of calculi. There was no diminution of the shadow following a fatty meal. This was considered contributory evidence of gallbladder disease.

June 8, 1937: Barium enema showed a normal large bowel. A long, slender appendix was seen in the right iliac fossa.

June 9, 1937: Duodenal drainage, after two stimulations by 50 cc. of 50 per cent magnesium sulphate solution, revealed no B bile and no cholesterol crystals or bilirubin pigment.

June 10, 1937: A second biliary drainage produced B bile which again failed to show cholesterol crystals or bilirubin pigment, but contained a moderate number of bile stained leukocytes.

June 17, 1937: A study of the small intestine showed a marked delay in the emptying of the stomach which contained a little barium up to the 48 hour period. There was also delay in the emptying of the second and third portions of the duodenum which appeared moderately dilated. This study was not diagnostic of definite pathology in the

small intestine. The roentgenologist stated, however, that such abnormal gastric and duodenal pathology could occur as a result of an acute cholecystitis.

June 14, 1937: Stool examination was negative for typhoid or dysentery organisms.

An attempt, June 21, 1937, to pass a tube into the duodenum was unsuccessful. About 50 cc. of barium were injected through it, however, into the stomach. This material passed readily through the pylorus into the duodenum and jejunum without delay, demonstrating the absence of obstruction in the third portion of the duodenum.

June 11, 1937: Gastro-intestinal series revealed a flattened superior surface of the duodenal bulb which was suggestive of periduodenal adhesions due to chronic cholecystitis. There was tenderness during this examination in the right upper quadrant and over the region of the gallbladder. No organic disease could be demonstrated in either the stomach or duodenum.

June 15, 1937: At 7.45 P.M. the patient vomited 900 cc. of cloudy material; at 3.45 A.M. she vomited 800 cc. more, and at 11.30 A.M., on June 16, 1,000 cc. of dark green fluid containing particles of undigested food.

A three position roentgenogram, at 10.30 A.M., June 16, failed to reveal evidence of acute intestinal obstruction. Her abdomen was soft, with slight, vague, indefinite tenderness in the upper portion of the right lower quadrant. There was no tenderness over the gallbladder region or in the right costovertebral angle.

A small suction tube was passed into the stomach through the nose and continuous suction was started. She was given a clysis of 1,500 cc. of Ringer's solution, and water by mouth was limited to not more than 30 cc. an hour. The tube was ejected repeatedly and had to be discontinued. In 24 hours, from 6/15/37 to 6/16/37 she vomited 2,200 cc. of olive-green fluid and 300 cc. had been obtained by the continuous suction tube.

Her temperature ranged between 99° to 100° F. Pulse varied from 60 to 80. Blood pressure remained at 150/100. The patient stated that she felt perfectly well, and at no time did she appear acutely ill. During this 48 hour period she had received 5,300 cc. of fluid, practically all of which was Ringer's solution and her output had been satisfactory. Urine was negative. White blood cells, 12,700; polymorphonuclears, 78 per cent. It was noteworthy that at no time did her abdomen show spasm, distention, palpable mass or viscera, and only very slight tenderness, from time to time. Her signs were essentially fleeting and inconclusive. Her sense of well-being and desire for food was of note.

At this time, in spite of the opinion of most of the surgical staff that she had a chronic gallbladder, it was felt that she had a partial, intermittent small intestine obstruction which the roentgenograms had failed to reveal. This attack subsided after three days. The leukocyte count had returned to normal by the nineteenth day and nasal gastric tube, which had been in place 72 hours, was draining clear bile and was removed. She had normal bowel movements with daily enemata during the attack. She improved steadily thereafter, but because of our inability to make a definite diagnosis it was thought justifiable to explore her abdomen.

Operation.—June 28, 1937: Avertin, gas-oxygen anesthesia. The gallbladder was found to be small, normal in color and without adhesions, it was not thickened, was easily emptied by digital pressure and contained no stones. The stomach, duodenum and pancreas were normal. The common duct felt normal. The omentum was densely adherent to the old, lower midline scar and a loop of ileum was attached to the lower end of the scar in a S formation. The adhesions between the antimesenteric border of the ileum and anterior abdominal wall had become stretched, to producing effect of a mesentery on the antimesenteric border. The site of the attached ileal loop was eight centimeters long. The ileum going up to and leaving the loop was sharply angulated at each end and was evidently the site of a partial recurring obstruction. The uterus, both tubes and ovaries were atrophic but normal. The appendix was normal.

The abdomen had been opened through a paracostal, right upper quadrant incision and upon finding no pathology in the biliary tract, a second midline incision, excising the old lower abdominal incision, was made and the ileum freed from the scar. The denuded surface of the ileum was peritonealized and both wounds closed in layers with silk.

The patient withstood the operative procedure very well and had an uncomplicated recovery, being discharged on the eighteenth day following operation.

Follow-Up.—Three months following operation, she stated that she felt well in every way. She had had no abdominal symptoms and a tablespoonful of mineral oil every morning effected a daily stool, which she had not had for ten years. She is free of all her preoperative symptoms, the wounds are firm, she has an excellent appetite and she has gained weight.

Comment.—This case is shown as an example of one of partial, recurrent intestinal obstruction due to postoperative peritoneal adhesions. The symptoms and signs at the time of admission, during an acute obstructive attack, were indefinite and confusing, simulating an acute exacerbation of a chronic cholecystitis, in a patient who had had typhoid fever seven years previously. She was seen by a number of the staff to whom the diagnosis was not clear and who even had doubts of the existence of the true pathology when seen during an acute attack of nausea, vomiting, abdominal pain and soft distention.

The three position roentgenograms, G. I. series, small intestine study, and barium enemata did not definitely show the true pathology. The fact that one duodenal drainage failed to show B bile, and a cholecystogram showed only a faint shadow with no emptying, confused the diagnosis. This type of case is dangerous because the diagnosis is difficult; however, in the presence of such symptoms, regardless of the laboratory data or physical examination findings, one must certainly consider the possibility of some degree of intestinal obstruction in view of the presence of an abdominal scar.

Discussion.—Dr. Seward Erdman (New York) remarked upon the marked reduction of mortality in the last four-year period. The several reports from the Presbyterian Hospital by Doctor vanBeuren now cover a period of several decades and constitute a very large and valuable series for analysis and comparison.

There is still lacking in medical literature any unanimity in the use of the adjectives applied to the word "ileus," such as dynamic, adynamic, postoperative, mechanical and paralytic; therefore, the simplicity of Doctor vanBeuren's two classifications, *i.e.*, (1) mechanical and (2) paralytic, is appreciated.

His personal belief was that the primary absence or presence of peritonitis was the chief difference between mechanical and paralytic ileus (which latter he prefers to call "peritonitic ileus"). Certainly all the enumerated types of mechanical ileus due to neoplasms, old adhesions and bands, strangulated herniae, gallstones, intussusception, volvulus, the obstruction, and some cases of wound disruption, occur, primarily, in a clean, noninfected abdomen, which as a result of delay in operating may become infected.

On the other hand in paralytic or "peritonitic ileus," some grade of peritonitis or at least some serious insult to the peritoneum by operation, precedes the obstructive symptoms. (The entire list of paralytic ileus cases in vanBeuren's series all had peritonitis.) But, almost universally, the "peritonitic ileus" soon develops a mechanical obstruction due to adherent loops and kinks, though it should still be classed as "peritonitic."

Some writers dignify the common 48 hour postoperative distention, pain, vomiting, and temporary paresis as postoperative paralytic *ileus*!—but certainly such cases when they yield to hot formentations, enemata, pitressin, and

stomach drainage, seldom deserve a classification as intestinal obstruction, or ileus.

Approaching ileus from the surgical standpoint, however, it will *be agreed* that, in all forms of mechanical obstruction, operation of one type or another is absolutely essential; and that lowered mortality will depend upon the promptness of surgical intervention, and the ability to deal with the underlying pathology. Of course, preliminary treatment to restore the body fluids and altered chemistry are essential.

Doctor vanBeuren states: "That in order to compare fairly the results in one series of ileus cases with another, each series must first be analyzed in three ways; (a) according to percentage of late cases; (b) according to cause or mechanism; (c) according to type—complete or incomplete, mechanical or paralytic."

He agreed absolutely with this statement and wished that all the carcinoma cases had been put into a separate classification because the mortality in such cases is much more dependent upon the underlying cancer than upon method of treatment.

Therefore, Doctor vanBeuren's Table IV showing the mortality of enterostomy in 39 patients, including a large number of colostomies for cancer, serves only to confuse the surgeon who would like to evaluate the results of enterostomies (of the small intestine) performed for the relief of acute ileus not complicated by cancer.

His interest in enterostomy for postoperative paralytic or "peritonitic ileus" began some 18 years ago and he had reported the earlier cases in the Surgical Clinics of North America in 1921, from the Second Surgical Division of the New York Hospital. In the next ten years, upwards of 70 high enterostomies were performed by members of that Staff for "peritonitic ileus" only too often without benefit, and sometimes without the best surgical judgment. Some six years ago, before the New York Surgical Society, he alluded to these cases and the late Dr. Frank S. Matthews, whose opinion he always regarded so highly, very properly criticized such indiscriminate use of enterostomy. He still believes, however, that enterostomy is a life saving measure in selected cases of postoperative "peritonitic ileus," but it is not easy to clearly define the indications in a given case or to select the most propitious time for employing this measure.

In the paralytic ileus resulting from a fulminating general peritonitis, it is of no advantage to perform an enterostomy, for it will not continue to drain. However, it is well recognized that a mechanical obstruction very often develops due to the matting together of intestinal loops by the peritonitis, and may prove to be the cause of death. It is in cases of "peritonitic ileus," with a mechanical obstruction added, that the brilliant results of a timely enterostomy are best demonstrated.

Mechanical obstruction following appendicitis requires some days to develop, probably from five to seven days, after the onset of the appendicitis; which, however, may mean at or soon after operation, in late operative cases.

"Peritonitic ileus" following appendicitis is so common, and the underlying pathology so nearly parallel in all cases, that a study of the results of enterostomy in this one disease would be most enlightening and perhaps in the future reports Doctor vanBeuren will segregate these cases. In the group of "Appendicitis with Fibrino-Purulent Peritonitis," Krech found the mortality ran between 57 and 93 per cent, and Dudley's statistics indicate an 83 per cent mortality, he also states that one-quarter of these die from mechanical obstruction. He believes that his own experience in 36 cases of enterostomy for "peritonitic ileus" after appendicitis, although showing a mortality over 50 per cent, nevertheless resulted in considerable saving of life.

ACUTE INTESTINAL OBSTRUCTION

A STATISTICAL SURVEY OF THREE HUNDRED FORTY-TWO CASES

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IN VIEW of the varying incidence of acute intestinal obstructions, especially of certain types of them, in different countries, a review on the occurrence of this condition in Japan might be of interest. The material which is considered deals with 342 consecutive cases operated upon by the author, between May, 1920, and July, 1935, in Kagoshima, situated in the farthest, southwestern part of Japan proper.

The cases are classified as follows:

(1) Intussusception.
(2) Volvulus.
(3) Strangulation by bands.
(4) Ileus by adhesion.
(5) Ileus caused by Meckel's diverticulum.
(6) Ileus due to internal hernia.
(7) Arterioduodenal ileus.
(8) Ileus by foreign bodies.
(9) Ileus by tumor.
(10) Compression ileus.
(11) Congenital intestinal obstruction.
(12) Spastic ileus.
(13) Ileus of unknown etiology.

Those cases which recovered after establishing a Witzel's fistula, which needed no further operation, or those which died after such an operation without improving, are classified as "ileus of unknown etiology."

Intussusception.—There were 154 cases with an operative mortality of 24 per cent. Intussusception is very common among the Anglo-Saxons. Although in Germany this is considered rare, according to the report by Ravens[24] of Kiel Clinic (Prof. Anschütz), intussusception proved to be the etiologic factor in 19 per cent in his 439 ileus cases (83 cases).

In Japan the statistics show 15 to 26 per cent of the whole number of cases of ileus to be caused by intussusception. Of the 342 cases upon which I have operated, 154 cases were found to be caused by intussusception (45 per cent). The frequency of occurrence of this condition in my series and that of the occurrence of volvulus in Russia, give practically the percentage, as in Russia intestinal obstructions are due to volvulus in almost 50 per cent of the cases.

It may be pointed out from my experiences that in Japan proper, intus-

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susceptions are very frequently encountered in the southwestern part of the country.

I am in accord with the opinion of many authors that the lack of fixity of the ascending colon to the posterior abdominal wall is a significant factor in the formation and development of intussusception. My opinion is based on the fact that in 38 of the 154 cases of intussusception, the ascending colon was found to lack its normal fusion to the posterior abdominal wall in its entire length, and in 39 cases, in its upper one-third or two-thirds.

In all statistics on intussusception the males have always predominated, and of my 154 cases, 108, or 70 per cent, were males, and 46, or 30 per cent, were females.

The relative frequency with respect to age was as follows (Table I):

TABLE I

AGE INCIDENCE

Age	Number of Cases	Percentage
0 up to 1 yr.	46	29.9
2 to 3 yrs.	23	14.9
4 to 10 yrs.	31	20.1
11 to 20 yrs.	13	8.4
21 to 30 yrs.	7	4.5
31 to 40 yrs.	14	9.1
41 to 50 yrs.	12	7.8
over 51 yrs.	8	5.2
Total	154	

In the present series, the youngest was a male, 30 days old, and the oldest was a male, age 67; both patients were cured by the operation.

The Starting Point of Intussusception varied as follows (Table II):

TABLE II

THE STARTING POINT OF INTUSSUSCEPTION

Starting Point	Cases	Percentage
Cecum	98	63.6
Lower portion of the ileum	49	31.7
Transverse colon	2	1.3
Ascending colon	1	0.7
Small intestine	4	2.6
Total	154	

Retrograde Intussusceptions have occurred in three instances. One of these was particularly remarkable, being retrograde, multiple, superimposed, and recurrent.

Case Report.—A male, age 20, was operated upon for an incarcerated inguinal hernia of the left side, August 23, 1925, 12 hours after the onset of the symptoms. As the incarcerated portion of the intestine was gangrenous, it was necessary to perform

a resection of that segment. The incarceration occurred 121 cm. above the ileocecal valve. Between the situation of the incarceration and the duodenojejunal flexure the intestine showed intussusceptions at ten different places, the uppermost one being situated 10 cm. below the duodenojejunal flexure. The distances between each distal intussusception were recorded as follows: Distances from the duodenojejunal flexure were, 10, 7, 35, 58, 42, 125, 48, 30, 32, and 27 cm. (Fig. 1).

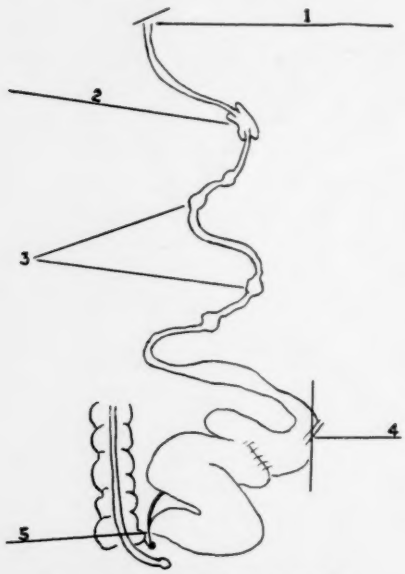

FIG. 1.—Showing the sites of multiple, retrograde and recurrent intussusceptions. (1) Duodenojejunal flexure. (2) Retrograde and downward invagination. (3) Invaginations at an early stage of their development. (4) Oblique fistula. (5) Obstruction from a band.

Most of these intussusceptions were retrograde and some of them were superimposed. They were reduced without difficulty, and the patient was discharged 16 days postoperative.

Recurrence.—Forty days after the first operation, October 1, 1925, at 10 A.M., the patient was suddenly seized with severe pain in the abdomen and four hours later a celiotomy was performed. The lower end of the ileum was found to be strangulated by a band and the intestinal wall of that portion was discolored. By removal of the band the intestinal passage was restored and the normal color of the intestine returned. At this time, multiple intussusceptions were again found in different portions of the small intestine, the uppermost one, 155 cm. below the duodenojejunal flexure, showing a retrograde, and usual (downward), intussusception at the same time. Four others were found in a very early stage of development of an invagination, each showing a ring-like fold of the intestinal wall (Fig. 1). They were all reduced.

Superimposed Intussusceptions are not common; six instances of them have been encountered.

Multiple Intussusceptions were found in the one case described above.

Symptoms of Intussusception showed the following frequence of appearance (Table III):

TABLE III

RELATIVE FREQUENCY OF APPEARANCE OF SYMPTOMS

Symptom	Percentage
Pain	91
Tumor	75
Vomiting	64
Passing of blood per rectum	60
Distention	34
Increased peristalsis	32

In four instances in this series the reduction took place spontaneously on the operating table before beginning the abdominal section. The children screamed and struggled desperately against the anesthetic and their very action evidently contributed to their recovery. In one of these cases the abdomen

was opened after the spontaneous reduction had apparently occurred, to ascertain whether the intussusception was completely disinvaginated or not. The celiotomy revealed only a small, hard, cup-shaped depression at the caput ceci.

Operative Mortality.—The shorter the interval between the onset of symptoms and operation, the lower the mortality will be. The operative mortality in this series was 24 per cent (Table IV).

TABLE IV

ANALYSIS OF AGE INCIDENCE AND OPERATIVE MORTALITY

Age	Cases	Cured	Died	Oper. Mortality Percentage
0 up to 1 yr.	46	34	12	26.0
2 to 3 yrs.	23	21	2	8.7
4 to 10 yrs.	31	25	6	19.3
11 to 20 yrs.	13	11	2	15.4
21 to 30 yrs.	7	5	2	28.6
31 to 40 yrs.	14	10	4	28.6
41 to 50 yrs.	12	7	5	41.7
over 51 yrs.	8	4	4	50.0

Anschütz warned against the opinion expressed by Perthes,[23] that the treatment of invagination should be conservative up to the age of one and one-half years, and urged the immediate surgical treatment in cases of acute invagination occurring in early childhood. Personally, I have not noted any particular danger in operating upon cases occurring in early childhood; on the other hand, the operative mortality, in my series, was much higher in the patients of advanced age (Table IV).

Resection.—According to Clubbe,[8] one must expect to find the bowel gangrenous or irreducible in about 6 per cent of the cases operated upon for intussusception, and in these cases one must employ resection. The mortality of various authors who have used this procedure has, so far, been very high. Of 154 cases upon which I have operated, resection had to be resorted to in 19 instances and six of them recovered. In my resection cases all of the successful results were obtained in patients more than five years of age.

Recurrence.—Clubbe experienced recurrence in two instances of his 253 intussusceptions, and in Close's[6] series (363 cases) intussusception occurred twice in 14 instances, and occurred three times in two cases. In my series intussusception occurred twice in three instances, and in one it occurred five times.

Prevention of Recurrence.—Clubbe warned against the procedure of anchoring the cecum as a means of lowering the mortality. To prevent the recurrence I have (when the condition of the patient permitted) fixed the cecum and ascending colon to the peritoneum of the right iliac fossa in 26 cases, after completion of the reduction. Of these 26 cases four died.

Volvulus.—Twenty-two cases; operative mortality, 27.3 per cent: To determine the frequency of volvulus in different countries, a survey was made by Muzeniek,[19] according to which, of the total ileus cases, volvulus was the

etiologic factor in 13 per cent in Germany, 16 per cent in France, and 12 per cent in America. Volvulus predominates in the acute intestinal obstructions of Russia. In Japan the statistics, by various authors on this subject, show variations of from 9 to 25 per cent. In my series volvulus constituted about 6 per cent of all ileus cases.

While in Europe males are more subject to volvulus than females, in Japan authors report that a majority of such cases are found in females. In my series, however, males predominated, the proportion being 13 to 9.

The sigmoid flexure is most frequently involved, the undue length of the bowel in this part being considered as a causative factor.

The sites of the volvulus in my series are as follows (Table V).

TABLE V

SITES OF VOLVULUS

Sigmoid flexure	14
Lower part of ileum	3
Small intestine	4
Ascending colon	1

Strangulation by Band.—Fifty cases; operative mortality 22 per cent.

Ileus by Adhesion.—Sixty-seven cases; operative mortality 31 per cent: In strangulation and ileus by adhesion in my series, appendicitis, past or present (37 cases), previous celiotomy (12 cases), and tubercular peritonitis (16 cases) were frequently found as the etiologic factor.

Ileus Caused by Meckel's Diverticulum.—Five cases; operative mortality, 60 per cent.

Ileus Due to Internal Hernia.—Four cases; operative mortality, 50 per cent: Of the four cases, one due to transmesocolic hernia, without sac proper, interested me as it is apparently very rare.

Case Report.—A male, age 74, was first seen July 4, 1926. There had been a bowel movement the previous day at 11 A.M. Thirty minutes later the patient complained of slight pain in the upper abdomen. It increased in intensity, and he vomited repeatedly. There was no flatus nor defecation after the onset of the pain. A prominent tumor-like, circumscribed distention was noted in the upper abdomen. The other part of the abdomen was comparatively retracted. The patient was operated upon at 5 P.M., July 4, 1926. There was some blood-stained fluid in the abdominal cavity. A coil of small intestine, 39 cm. long, was festooned in the shape of a horse-shoe over the anterior wall of the stomach, through the openings in the mesocolon and the small omentum. The construction of the bowel at the opening in the mesocolon caused the acute intestinal obstruction. The ensnared coil was distended and slightly discolored. The upper end of the involved intestine was found 31 cm. below the duodenojejunal flexure. The stomach was slightly distended. After reduction the abnormal openings were closed by sutures. Recovery was uneventful (Fig. 2).

Arterio-duodenal Ileus.—Three cases; operative mortality, 66.7 per cent: In all three instances the small intestines had dropped down into the pelvis, stretching the superior mesenteric artery and vein, so that they compressed the duodenum and obstructed it. As the intestines were lifted up, the pressure on the duodenum was relieved and the lumen of the obstructed gut was re-

stored; but on allowing them to drop free the compression again occurred. A gastro-enterostomy was, therefore, performed in each instance.

Ileus by Foreign Bodies.—Eight cases; operative mortality, 60 per cent. (Ascaris, four cases; inspissated feces, one case; others, three cases.)

Ileus by Tumor.—Six cases; operative mortality, 66.7 per cent.

Compression Ileus.—Three cases; operative mortality, 33 per cent.

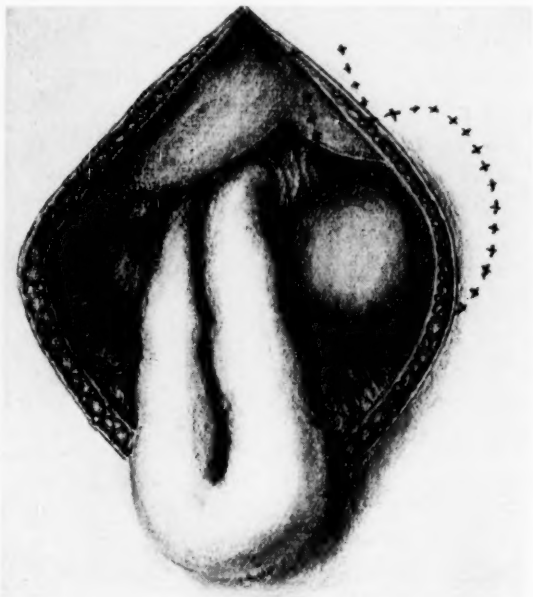

FIG. 2.—Showing the appearance of the strangulated loop of intestine in the case of transmesocolic hernia.

Congenital Intestinal Obstruction.—One case; operative mortality, 100 per cent.

Case Report.—Male, age three days. Soon after birth the baby began to vomit frequently. A rectal irrigation resulted in the passage of some mucus. When first seen, December 15, 1927, the abdomen was extremely distended and showed increased peristalsis. The baby looked very anxious and screamed at short intervals. Upon operating, a small amount of fluid was found in the abdominal cavity. The first obstruction was found 93 cm. below the duodenojejunal flexure, and the second, 10 cm. below the first; both were due to a congenital anomaly. The stomach and intestines, down to the first obstruction, were extremely distended. An oblique fistula was made above the first obstruction, in order to relieve the distention; and for the second, an enterostomy was performed. Death ensued 12 hours postoperative.

Spastic Ileus.—One case; operative mortality, zero.

Symptoms.—The frequency of appearance of symptoms in the present series is as follows (except intussusception) (Table VI).

Operative Mortality.—In spite of the progress of surgery in the last few decades the operative mortality of acute intestinal obstruction is still too high. The mortality percentage depends upon the time elapsed and the ability of

TABLE VI

FREQUENCY OF APPEARANCE OF SYMPTOMS

Pain in the abdomen	93%
Vomiting	78%
Constipation	70%
Distention	65%
Increased peristalsis	65%
Rumble	51%

the surgeon; that is, the shorter the time between the onset of the symptoms and the beginning of the surgical procedure, the lower will be the mortality rate. Of 342 cases operated upon, the mortality was 32 per cent. If the intussusceptions are excluded, the mortality of the remaining 188 cases rises to 38.8 per cent.

Results of Operating at Various Stages.—The idea expressed in the last paragraph can perhaps be better shown, and its importance more clearly grasped, from Table VII (intussusceptions excluded):

TABLE VII

MORTALITY RESULTING FROM OPERATING AT VARIOUS STAGES

Interval between the Onset of Symptoms and the Operation	Cases	Died	Operative Mortality Percentage
Up to 12 hrs.	21	5	23.8
13 to 24 hrs.	29	9	31.0
25 to 48 hrs.	48	15	31.0
Over 49 hrs.	90	44	49.0

Treatment.—The general consensus of opinion of many surgeons is that the surgical procedure applied to acute intestinal obstruction must be minimized, for one must bear in mind the impoverished resistance of these patients to surgical interference. In the presence of marked distention it is impossible to definitely disclose the pathologic conditions within the abdomen. If one operates upon such a case radically, in one stage, there is great danger of damaging the friable intestinal wall and contaminating the abdominal cavity with the intestinal contents.

When circumstances do not permit the radical operation, an oblique fecal fistula has been placed in the most distended coil, and a small rubber tube introduced into the gut through a small incision. I strongly warn against the formation of a simple fecal fistula, because the subsequent treatment for the fistula is an annoying one. The oblique fistula never produces a permanent artificial anus. Later, when the distention has been reduced by drainage through the fistula and the critical period has been passed, the abdomen should be widely opened, and the cause of the obstruction removed. Fortunately, the most distended portion of the coil, where the oblique fistula was formed, is always found to be just above the obstruction. It may be considered an excellent indication, if the intestinal content begins to drain

through it at the moment, or soon after its formation. This drainage through the fistula does not begin, unless the peristaltic movement of the bowel is restored. It is not rare that flatus and feces per rectum will be evacuated without further operation after the formation of the fistula. If the bowel begins to move normally, the drainage through the fistula stops spontaneously. The rubber tube is then removed and the fistula heals spontaneously.

An oblique fistula was effected in 72 instances, and 40 patients were saved by this procedure (operative mortality, 44 per cent). This mortality rate may appear too high, but it must be taken into consideration that this procedure was used more especially in very serious cases, where the surgical risk with more complicated manipulations would have been correspondingly greater.

In the present series there are 15 instances where, after the formation of the fistula, no further operation was necessary. In 27 cases subsequent operations were needed. The average interval between the formation of the fistula and the subsequent operation was 17 days.

Ileus Caused by the Oblique Fistula.—There were five cases of acute intestinal obstruction caused by the kinking of the coil at the point where the oblique fistula had been previously formed. However, it is relatively an easy matter to release the kink by operation and no deaths resulted from the condition.

Operative Closure of the Transverse Fecal Fistula in the Early Stage.—In some cases of acute intestinal obstruction due to strangulation, especially from volvulus, the blood circulation of the mesentery is so greatly interfered with that the bowel becomes gangrenous in a remarkably short time. The patient collapses and suffers profoundly from toxemia. Upon opening the abdomen, blood-stained fluid escapes and the gangrenous loop, a foul-smelling and discolored mass, is found. In such circumstances the surgeon must attempt to save the patient by the simplest and speediest procedure possible, namely, that of drawing out the gangrenous loop and resecting it. However, difficulties follow the employment of this procedure, because dehydration, irritation and soiling of the surrounding skin by discharge from the fecal fistula, and inanition, when the fistula is formed high in the small intestine, cannot be avoided. As a rule it requires a long time to perform the second operation for the closure of the fistula, as the chances of recovery depend largely upon the condition of the affected skin surrounding the fistula.

For shortening the time between the formation of the fistula and the second operation, a consideration of Tschmarke's[27] technic will be found helpful. I have operated twice in the following manner:

After resection of the gangrenous loop, rubber tubes, 25 cm. long, are put into each of the cut ends of the bowel, and held in place by tying a string around the outside of the bowel. The two cut ends of the bowel are thus brought out through the incision, side by side, and their serous surfaces are sutured to the parietal wound, allowing about 2 cm. of each cut end to project beyond the surface of the skin. The two ends of the bowel outside the peritoneal cavity are surrounded with gauze. During the next few days the content of the

bowel drains through the rubber tube without soiling the wound or skin, since the two external ends of the tubes are deflected to the side of the abdomen.

The second operation is very simple. By removing the sutures, the abdomen is reopened, and the two ends of the bowel, which have been kept outside, are resected and united end-to-end. The second operation must be performed as soon as possible, as the bowel bound to the rubber tube soon becomes gangrenous and leakage of the bowel content occurs.

ILLUSTRATIVE CASE REPORTS

Case 1.—Volvulus of the Small Intestine: Male, age 31. At the first operation 240 cm. of the involved gangrenous loop were resected and treated by the above mentioned procedure. The second operation for the closure of the fecal fistula was undertaken three days later. Since the third day after the second operation, the bowel has moved normally, and the result of the operation itself was excellent. But, unfortunately, the patient succumbed as the result of cerebral embolism.

Case 2.—Volvulus of the Ascending Colon and the Lower End of the Ileum: Female, age 42. At the first operation the gangrenous loop was resected and treated as above mentioned. The second operation for the closure of the fecal fistula was undertaken two days later. Normal bowel movement followed the second operation, and the patient was enjoying an uncomplicated convalescence, when she contracted acute pneumonia, from which she died seven days after the second operation.

SUMMARY AND CONCLUSIONS

(1) A statistical survey of 342 cases of acute intestinal obstruction operated upon during the last 15 years and 3 months is made.

(2) Intussusceptions constituted 45 per cent of all ileus cases.

(3) The partial or total lack of fixity of the ascending colon to the posterior abdominal wall is considered as a significant factor in the formation and development of intussusception. Of the 154 cases of intussusception, it was found that in 38 cases the ascending colon lacked its normal fusion to the posterior abdominal wall in its entire length, and in 39 cases in its upper first or second third.

(4) The average operative mortality of all of the series was 32 per cent; in the cases of intussusception exclusively, it was 24 per cent.

(5) Although it is generally believed that the operative mortality following intussusception is very high in children under the age of one year, it is not true in the writer's case; on the other hand, the operative result is much more markedly unfortunate in the patients over 40 years of age.

(6) The factors which may reduce the operative mortality, are early diagnosis and immediate surgical interference in the earliest stage of the disease.

(7) In serious cases of acute intestinal obstruction, surgical procedures must be reduced to the minimum. For this reason the writer has frequently employed that of effecting an oblique fistula. The simple, direct intestinal fistula results in a permanent fecal fistula. This complication can be prevented by the formation of an oblique fistula, using a thin rubber tube.

(8) A special method of procedure is suggested which permits the earliest possible closure of the transverse intestinal fistula.

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PERFORATED GASTRIC AND DUODENAL ULCER *

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ACUTE perforated ulcers of the stomach and duodenum still compose one of the most dramatic of the acute abdominal surgical conditions. They are of interest to the physician, who usually makes the diagnosis; to the surgeon who—in his decision as to the character of the operative procedure to be carried out—must be guided not only by the limitations implied by the patient's condition, but also by his own limitations, since these cases are emergencies often cared for by the junior members of the attending staff; and to both in the effect of perforation and procedure upon the underlying condition.

A review of recent literature shows that there is still considerable difference of opinion as to the most advisable procedure—ranging from closure alone[1, 2, 3, 4] to—chiefly from German and Russian sources[5]—a large percentage of immediate resections. Drainage[2, 6, 7] is "always," "sometimes," or "never" advised.

With so much variation of opinion, standardization of procedure is still to be determined, and it, therefore, seems proper to present the experience of a single service, extending over a 16-year period. A small group of the earlier cases was reported in 1923.[8] This group has been included in the present series.

One hundred seventy patients with acute perforated peptic ulcer were admitted to the First Surgical Division of Bellevue Hospital between January 1, 1920, and December 31, 1936. Of these, 162, or 95 per cent, were male. Table I shows the age incidence of these cases.

TABLE I

AGE INCIDENCE*

Decade	Cases
11–20	3
21–30	37
31–40	40
41–50	34
51–60	33
61–70	12
71–80	2
81–	1
Total	162

* Eight were women, four between 31–40, four between 41–50.

* Read before the Association of the Alumni of Bellevue Hospital, New York, May 5, 1937. Submitted for publication August 16, 1937.

Perforated ulcer is peculiarly a disease of the male sex and the number of women reported here is larger than those reported in similar series. It may be that increased abdominal tension associated with more strenuous occupation has something to do with this difference in incidence. At least we believe that increased pressure from the outside has something to do with hurrying an impending perforation, having seen several cases perforate shortly after fluoroscopy or after too thorough examination by a group of students.

History.—The previous history is of little importance. One hundred nine cases, or 64 per cent, gave a history suggestive of ulcer over periods of from one month to 18 years. Those with gastric ulcers gave longer histories than those with duodenal ulcers. Thirty cases, or 18.5 per cent, had had indefinite indigestion for from two weeks to ten years. Thirty-one cases, or 19.1 per cent, had had no previous digestive symptoms, the perforation being the first evidence of disease. Five patients had had hematemesis, 13 reported tarry stools, and 20 reported loss of weight up to 40 pounds.

Poor economic conditions may be a factor in the frequency of perforations, although there has not been a marked difference in our figures during the past eight years—no more than would be accounted for by the increased demand on a city institution. On the other hand, Zukschwerdt and Eck,[9] reporting a series of 117 cases from Heidelberg in 1931, felt that there had been a distinct increase in their cases since 1924, which they thought to be due to improved nutritional conditions.

A surprisingly small number of our cases had had previous treatment. Twenty-seven had had medical treatment of varying duration and thoroughness; ten had had "chronic appendices" removed with no resultant improvement; and five had had previous perforations. In three of these, simple closures had been effected, but it was not possible to determine whether or not the second perforation was at the same site. In the others gastro-enterostomies had been added to closure of the perforations, and in both of these the second perforations were in the jejunum: one in the proximal loop, the other in the distal. One patient had been given osteopathic treatment following the perforation.

Symptoms and Physical Signs.—These have been described so often that it seems necessary to comment only on the presence or absence of shock. We believe that this has been given a much more prominent place in the textbooks than is warranted. Shock probably depends upon the size of the perforation and the amount and rapidity of flow of intestinal contents into the peritoneal cavity. It was not noted in four cases in which perforation occurred in patients under observation. The relative infrequency of shock is suggested by the blood pressure readings on admission. In 63 cases which recovered, this varied from 95/60 in a case admitted ten hours after perforation to 170/105 in a patient, age 63, admitted five hours after perforation. In only one case was the systolic pressure below 100. In 19 fatal cases the systolic pressure was under 100 in seven, six of which had perforations of over 24 hours' standing.

The temperature and pulse are of comparatively little value. The temperature on admission was over 100°F. in 22 of 134 cases which recovered, and in 12 of 36 fatalities, while the pulse rate was over 100 in 20 cases in each group.

Blood counts in the recovered group varied from 3,000, with 86 per cent polymorphonuclears (four hours postperforation) to 29,900, with 85 per cent polymorphonuclears (four hours postperforation), while in the fatal cases the limits were 3,000, with 90 per cent polymorphonuclears (29 hours) and 24,000, with 96 per cent polymorphonuclears (30 hours) (Table II).

TABLE II

WHITE BLOOD COUNTS

Recoveries	Cases	Deaths
Under 10,000 W.B.C.	28	6
10,000–20,000 W.B.C.	69	19
Over 20,000 W.B.C.	21	5

We have made very little use of roentgenologic examinations for diagnosis.[11, 12] The symptoms and signs are usually so clear-cut, and the diagnosis so definite, that the delay and, more important, the moving and manipulation of the patient are unnecessary.

Finsterbruch and Gross have collected and reported 146 cases in which roentgenologic examination has been used for diagnosis, with 95 per cent correct results. They emphasize, however, the fact that the absence of pneumoperitoneum, as shown by the film, does not eliminate the possibility of a perforation being present.

TABLE III

PATHOLOGY

Duodenal Ulcers	54		
Indurated	36	Soft	18
Sealed	19	Open	35
Peritoneal Contamination			
None	18		
Moderate	11		
Extensive	23		
Abscess	2		
Gastric Ulcers	109		
Indurated	79	Soft	30
Sealed	24	Open	85
Peritoneal Contamination			
None	20		
Moderate	30		
Extensive	58		
Abscess	1		
Jejunal Ulcers	2[13]		
1 in proximal loop, with acute diffuse peritonitis			
1 in distal loop, with acute diffuse peritonitis			
Undetermined by operation or autopsy—5			

The figures in Table III are open to question because of the difficulty of determining the exact relation of a pyloric perforation to the pyloric vein. Forty-five cases were described in the operator's report as "pyloric, probably gastric" or "duodenal" as the case might be. These cases have been grouped with those definitely gastric or duodenal for the sake of simplicity.

With two exceptions all the perforations were on the anterior wall. Two perforated into the lesser sac. Both of these were indurated ulcers, open at the time of operation, with extensive contamination of the general peritoneal cavity through the foramen of Winslow. Repair of the perforation in these cases is necessarily difficult and unsatisfactory.

TABLE IV

BACTERIOLOGY

Cultures taken in 42 cases

20 Sterile (2 over 9 hrs. after perforation)
- 14 Primary union
- 2 Wound infection
- 2 Disruption
- 2 Died before wound required dressing

22 Positive cultures* (8 under 6 hrs. after perforation)
- 7 Primary union
- 9 Wound infection
- 2 Disruption
- 4 Early deaths

* The cultures in these cases were almost invariably Streptococcus and Staphylococcus. Colon bacilli were found only in the late cases.

Wilensky,[12] in 1922, suggested that the presence or absence of induration about a perforation might be an indication of the necessity of more extensive procedures than a simple closure—"not necessarily at the time of the perforation." This has not been borne out by our experience in the follow-up; soft and indurated ulcers requiring postoperative care and secondary operation in about the same percentage of cases.

TABLE V

PREOPERATIVE DIAGNOSES

These were correct in all but eight cases, namely:
- 6 Acute appendicitis
- 1 Acute diffuse peritonitis (cause unknown)
- 1 Bleeding gastric ulcer*

* In the last case hemorrhage and perforation were apparently simultaneous, and the perforation was not suspected, being found at autopsy.

The analysis of the operative procedures in Table VI would tend to indicate our preference for conservative measures—especially if one realizes that most of the gastro-enterostomies were performed during the early years considered in this analysis. At present it is our belief that closure alone is suffi-

cient, with the rare exception (one in 170 cases in this series) in which extensive induration makes closure impossible, while early operation makes more radical procedure—gastrectomy—justifiable.

Obstruction following closure is rare, the edema and infiltration disappearing rapidly, so that immediate gastro-enterostomy is very rarely necessary and early secondary operation unusual.

Drainage.—We very rarely drain the peritoneal cavity, believing that with closure of the perforation and mechanical cleansing of the peritoneum, the infectious process will be adequately controlled by the individual. When secondary abscesses develop—usually subphrenic or subhepatic—they are cared for as may be necessary.

TABLE VI

OPERATIVE PROCEDURES

Perforated Duodenal Ulcer
- 36 Duodenorrhaphy
- 15 Duodenorrhaphy and posterior gastro-enterostomy
- 3 Duodenorrhaphy and appendicectomy
- 1 Drainage of subphrenic abscess

Perforated Gastric Ulcer
- 87 Gastrorrhaphy
- 7 Gastrorrhaphy and posterior gastro-enterostomy
- 2 Gastrorrhaphy and appendicectomy
- 1 Gastrorrhaphy and pyloroplasty
- 1 Gastrorrhaphy and ligation of lesser curvature vessels
- 1 Gastrorrhaphy and jejunostomy
- 1 Partial gastrectomy
- 1 Appendicectomy (perforation firmly sealed)
- 1 Jejunostomy
- 1 Gastrostomy
- 1 Gastrostomy for posterior perforation
- 2 Drainage of peritoneal cavity

Perforated Jejunal Ulcer
- 2 Jejunorrhaphy

No operation—8

In eight cases no operative procedure was carried out. Two of these patients were moribund and in one the perforation was found only at autopsy. The other five patients recovered. All of these came to the hospital over 24 hours after perforation, with minimal symptoms and physical signs. In all the diagnosis was confirmed by roentgenologic examinations which showed free air between the liver and diaphragm.

It is our belief that this is not an uncommon event. A small perforation occurs at a time when the stomach is empty and is immediately sealed over by omentum or liver. If the patient comes under observation early, the signs of peritoneal irritation are marked enough to warrant operation, which reveals a minimal amount of contamination and a perforation already sealed off. This picture—minus the signs of peritoneal irritation—is not infrequently seen in interval operations for ulcer. Later questioning of these patients may

recall to mind an attack of pain more severe than usual, possibly accompanied by abdominal soreness, but gradually subsiding.

TABLE VII

SECONDARY OPERATIONS DURING POSTOPERATIVE STAY IN HOSPITAL

Number of Operations	Nature of Procedure
4	Secondary suture of abdominal wall
3	Jejunostomy
2	Drainage of subphrenic abscess
3	Posterior gastro-enterostomy

Postoperative complications, aside from intraperitoneal abscesses—usually subhepatic or subphrenic—occurred chiefly in the respiratory organs (Table VIII). Their frequency is indicated in the following table:

TABLE VIII

POSTOPERATIVE COMPLICATIONS

	Recoveries	Deaths
Pneumonia	22	7
Pulmonary embolus	1	1
Bronchitis	5	
Pulmonary tuberculosis (probably antecedent)	2	

Table IX shows the incidence and nature of the occurrence of nonpulmonary, postoperative complications.

TABLE IX

NONPULMONARY, POSTOPERATIVE COMPLICATIONS

	Recoveries	Deaths
Gastric fistula	1	1
Duodenal fistula		1
Cerebral hemorrhage		1
Acute parotitis	2	
Disruption of wound	4	1

Mortality.—As in all reported series, the mortality percentage depends almost entirely upon the length of time between the perforation and operation. The total mortality in our series was 20.5 per cent—35 deaths in 170 admissions (Table X).

TABLE X

ANALYSIS OF THE 35 PATIENTS THAT DIED

Time between Perforation and Operation	Recoveries	Deaths	Mortality Percentage
1– 6 hrs.	87	4	4.3
6–12 hrs.	32	8	20
12–24 hrs.	9	5	35.7
24–48 hrs.	3	12	80
48 hrs. and later	2	3	60
No operations	5	3	37.5

Fifteen patients died within 24 hours of operation. These were hopeless cases in which operation might well have been refused, but in which it was performed with the feeling that an open perforation was present and that closure offered the patient his only chance.

In the three fatal cases in which no operation was performed, two were moribund on admission, while in one massive hemorrhage and perforation occurred simultaneously and the perforation was recognized only at autopsy.

There were 17 later deaths (Table XI).

TABLE XI

ANALYSIS OF 17 DEATHS OCCURRING AFTER 48 HRS. POSTOPERATIVE

Time of Death	Operation	Cause of Death
3rd day	Gastrorrhaphy	Pulmonary embolus
3rd day	Gastrorrhaphy	Pneumonia
4th day (2 cases)	Gastrorrhaphy	Pneumonia
4th day	Gastrorrhaphy	Acute diffuse peritonitis
5th day	Gastrorrhaphy and pyloroplasty	Acute diffuse peritonitis
5th day	Gastrorrhaphy	Acute diffuse peritonitis and pneumonia
5th day	Gastrorrhaphy	Acute diffuse peritonitis and gastric fistula
7th day	Gastrorrhaphy; secondary jejunostomy	Acute diffuse peritonitis
9th day	Gastrorrhaphy	Acute diffuse peritonitis
10th day	Duodenorrhaphy	Subphrenic abscess; fistula
10th day	Duodenorrhaphy	Hemorrhage
12th day	Gastrorrhaphy; secondary jejunostomy	Acute diffuse peritonitis and pneumonia
13th day	Gastrorrhaphy	Cardiac failure
16th day	Gastrorrhaphy	Secondary perforation
31st day	Gastrorrhaphy and ligation of arteries	Pneumonia
36th day	Jejunorrhaphy	Plastic peritonitis

Follow-Up Results: Follow-up in a municipal hospital service is difficult because of the constant shifting of the population—a shift usually carried out without notice to the postal authorities. However, we have been able to follow, for varying periods of time, 118 of 135 discharged patients or 87 per cent (Table XII).

Two of these cases came back after ten years or more of complete relief following simple closure of a perforation, with definite ulcer symptoms. In these cases we believe that a new ulcer had developed, possibly at the site of a previous perforation, rather than that the ulcer had been persistent, though quiescent, for many years. These recurrences, after a long period of time, emphasize how difficult it is to close the "follow-up" of such cases in the belief that the final stage has been reached.

SUMMARY.—A series of 170 acute, perforated peptic ulcers treated on the First Surgical Division of Bellevue Hospital, between 1920 and 1936, is presented.

TABLE XII

RÉSUMÉ OF FOLLOW-UP RESULTS

		Time Followed	Result
DUODENAL ULCER			
29	Closure		
17		2– 90 mos. (average 34 mos.)	Complete relief
5		6– 34 mos. (average 21 mos.)	Occasional gas after over-eating
7*		1– 41 mos. (average 19 mos.)	Recurrent ulcer
14	Closure and Posterior Gastro-enterostomy		
11		2– 9 mos. (average 42 mos.)	Complete relief
1		12 mos.	Occasional gas after over-eating
2		23–132 mos.	Recurrent ulcer
GASTRIC ULCER			
61	Closure		
39		2– 93 mos. (average 26 mos.)	Complete relief
9		3– 42 mos. (average 23 mos.)	Occasional gas after over-eating
13†		1–123 mos. (average 20 mos.)	Recurrent ulcer
6	Closure and Posterior Gastro-enterostomy		
4		16 –95 mos. (average 58 mos.)	Complete relief
1		12 mos.	Occasional gas after over-eating
1‡		63 mos.	Recurrent ulcer
No Operation			
3			
1		16 mos.	Complete relief
1		12 mos.	Occasional gas after over-eating
1		2 mos.	Persistent ulcer—Posterior gastro-enterostomy with complete relief—42 mos.
Miscellaneous Operations			
5	1 Gastrectomy	72 mos.	Complete relief
	1 Jejunorrhaphy	28 mos.	Complete relief
	1 Drainage of sub-phrenic abscess	32 mos.	Complete relief
	1 Appendicectomy (sealed perforation)	4 mos.	Complete relief
	1 Jejunostomy (sealed perforation, acute diffuse peritonitis)	50 mos.	Occasional discomfort

* Two of these cases have had secondary posterior gastro-enterostomies performed, and have been free from symptoms for 90 and 100 mos. respectively. The others have refused further operation.

† Eight of these patients underwent secondary operations. One had an exploratory celiotomy—no lesion found. Three had posterior gastro-enterostomies, of which group one was lost from follow-up; the second required revision after 48 mos.; and the third had complete relief for 33 mos. Four had partial gastrectomies with complete relief for 3, 15, 33 and 60 mos., respectively.

‡ This patient underwent partial gastrectomy with complete relief (30 mos.).

The predominance of perforations on the anterior surface of the stomach and duodenum is emphasized in this series, as is the relative infrequency of this condition in the female.

The importance of early diagnosis and operation is again stressed by our mortality percentages.

The difficulty in determining the final result is shown by the cases returning in ten or more years after perforation with recurrence of symptoms.

CONCLUSIONS

While the percentage results in the follow-up of a few cases would tend to show that closure plus gastro-enterostomy gave better results, it is our belief that closure alone is the more satisfactory procedure; the alternative in occasional cases with extensive induration making closure difficult—when the patient's condition justifies it—being a partial gastrectomy.

Closure alone may necessitate more secondary operations, but will lessen the number of serious secondary procedures such as secondary gastrectomies for marginal ulcer, a procedure which taxes the skill of the most experienced surgeon as well as the resistance of the best prepared patient.

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MESENTERIC LYMPHADENITIS AND THE ACUTE ABDOMEN

REPORT OF THIRTEEN CASES

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INFLAMMATION of the mesenteric lymph nodes was recognized and discussed as early as the eighteenth century. The diagnosis was usually made after the patient came to autopsy and descriptions of cases were confined chiefly to necropsy examinations. Enlarged mesenteric lymph nodes, discrete or matted together, were found showing various degrees of caseation, suppuration, or calcification. These were generally assumed to be caused by tuberculosis. The term "tabes mesenterica," given to this condition in 1775, by Ball, is still used. Tuberculous mesenteric lymphadenitis, as a surgical entity, had received so little attention at the beginning of the twentieth century that Mächtle found only 14 cases in the literature in 1909.

With the advent of abdominal surgery an increasing number of cases were reported in which an enlargement of the mesenteric lymph nodes was the only finding which explained the clinical syndrome of an acute abdominal condition. The lymphadenopathy was commonly thought to be caused by the tubercle bacillus, although many cases revealed only moderate enlargement of the lymph nodes with no matting, caseation, suppuration, or calcification. With the development of the Mantoux test and improvement in culture methods there accumulated cases with negative tuberculin reactions having pyogenic organisms in enlarged nodes. These cases established nontuberculous mesenteric lymphadenitis as a definite clinical entity. Some observers, at present, believe that the tuberculous and nontuberculous adenitis may be different phases of the same disease. Because mesenteric lymphadenitis may simulate numerous acute, surgical, abdominal conditions; because it is not uncommon, and because many of its features are not yet fully understood, observations on a series of cases are herewith presented. In eight of the 13 cases reported, a positive diagnosis was made at operation. In the remaining five a *presumptive* diagnosis of mesenteric lymphadenitis was made, a decision against operation being justifiable in view of our previous experience.

CASE REPORTS

Case 1.—No. 68665. R. S., male, age five, was admitted to the hospital on the evening of July 8, 1933, complaining of severe epigastric pain of two days' duration. The parents stated that the child had been well until, two days before admission, he complained of a pain in the left upper quadrant of the abdomen, which was transitory but severe enough to double him up. He had a similar attack the evening of the same day and was given castor oil. He felt well from then until, on the morning of admission, he complained of

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slight pain in the stomach. This persisted and was mild in character. At noon his mother gave him an enema and he felt relieved. An hour later he again had very severe pain which subsided but recurred after an hour. It was then situated in the region of the umbilicus and caused the patient to cry. He was nauseated but did not vomit. There was a past history of "trouble with enlarged tonsils and adenoids."

Physical Examination revealed generalized rigidity over the abdomen without localized tenderness. The heart and lungs were negative. Rectal temperature 38.3° C.; white count 15,800. The urine was negative. Roentgenologic study of the abdomen revealed no radiopaque areas within the chest or abdomen. A Mantoux test with 1 : 1,000 tuberculin was positive. A diagnosis of acute appendicitis could not be ruled out and an exploratory celiotomy was advised.

Operation.—The appendix was examined through a right rectus incision. It was found to be longer and larger than one would expect in a child of five but was not inflamed. It was removed and the stump invaginated. There was no Meckel's diverticulum and the bowel wall appeared normal. The mesentery, especially at its base near the terminal ileum, contained a large number of swollen lymph nodes. Enlarged nodes were also present in other parts of the mesentery. The symptoms were attributed to the mesenteric adenitis. The cause for the inflammation of the nodes could not be determined. The wound was closed without drainage.

The patient had a very mild convalescence and the wound healed by primary intention. The temperature dropped from 38.5° to 37.8° C. within 24 hours and remained at that level or lower during the nine days of hospitalization. The patient has had no further symptoms during the 14 months since the operation.

Case 2.—No. 107410. T. G., male, age one year, was first seen July 4, 1934, for a complaint of periodic cramping abdominal pains of 24 hours' duration. An accurate history was difficult to obtain because of the parent's lack of intelligent observation. The child was admitted for emergency treatment. He had been perfectly well until 24 hours previously when he began to vomit "yellow material" and to have periodic abdominal cramps. About six hours before admission the vomiting and pain became very severe. There was no history of bloody stools. The child had been constipated for 36 hours but an enema had given no result.

Physical Examination revealed a pale, very acutely ill child with grunting respirations. Temperature 37.5° C. The heart and lungs were normal. The abdomen was fairly soft and a mass was palpated in the right lower quadrant, on the bimanual examination. No blood was found on rectal examination. The white cell count was 41,500. Intussusception in the region of the ileocecal valve was suspected and operation was advised. The patient was given 500 cc. of Ringer's solution containing 25 Gm. of glucose subcutaneously.

Operation.—Under local anesthesia, a right, midrectus incision was made. The mass palpated before operation was an enlarged right kidney lying in the region of the right iliac fossa. The small and large intestines were of normal size and color. The appendix appeared normal. The mesentery of the ileum was studded with enlarged lymph nodes, ranging from 1/3 to 1½ cm. in diameter. The appendix was removed and the wound was closed without drainage. The child's condition appeared unchanged for about four hours, when he suddenly became worse. The temperature rose to 39° C., and the child expired five and one-half hours after operation. A postmortem examination was refused.

Case 3.—No. 91730. E. G., female, age seven, was seen November 13, 1933, for a complaint of pain in the region of the umbilicus, and vomiting. About three months before admission, while at school, the child vomited and developed pain and tenderness in the region of the epigastrium. This attack was of very short duration (about one day) and no more symptoms presented until about one month later, when she had a similar attack, and a third one month before admission. At that time her temperature was 38.9° C.; white count 28,000. There was slight tenderness about the umbilicus; otherwise

the physical examination was negative. Her white count dropped to 12,000 within three days.

The present attack began November 12, 1933, when she awoke complaining of pain and tenderness in the region of the umbilicus. She vomited once at that time. Temperature on admission 38° C. The white count was 5,600.

Physical Examination revealed subacutely inflamed tonsils, and there was a faint systolic murmur at the apex of the heart. There was some tenderness about the umbilicus, but there was no distention. There were no palpable masses in the abdomen. A definite diagnosis could not be made. However, a recurring appendicitis or a Meckel's diverticulitis was considered. After the administration of fluids the white cell count was 10,800. Roentgenologic examination of the entire G. I. tract was normal. Four days after admission, operation was advised.

Operation.—The abdomen was opened through a lower right rectus incision. The appendix appeared entirely normal. There was no evidence of a Meckel's diverticulum. Eight or ten enlarged lymph nodes were found in the mesentery at the ileocecal junction, and many more were found in the mesentery of the ileum higher up. These nodes were very light pink in color with a granular appearing surface. Two were removed for culture and microscopic examination. The appendix was removed and the wound was closed without drainage.

Microscopic Examination of the appendix revealed marked lymphoid hyperplasia but no evidence of inflammation. Culture of the mesenteric lymph nodes showed *Streptococcus viridans.* No signs of tuberculosis developed in a guinea-pig inoculated with material from the nodes. A Mantoux test with 1 : 100 tuberculin showed no reaction. The child made an uneventful recovery and has had no recurrence of symptoms.

Case 4.—No. 110514. S. W., male, age nine, was admitted to the hospital August 17, 1934, with a complaint of vomiting for the preceding three and one-half hours. He stated that the night before he had been "sick to his stomach." He had been given an "Ex-Lax" tablet and had gone to sleep but awakened at midnight with pain about his umbilicus. The following morning he began to vomit and continued to vomit for about three and one-half hours. He had taken nothing by mouth since that time. The bowels had been regular and there had been no urinary complaints. The past history was irrelevant.

Physical Examination.—The patient was well developed and well nourished. Rectal temperature 38.2° C. The tonsils were small and definitely inflamed. There was localized tenderness over McBurney's point which radiated to the umbilicus. Rectal examination was negative. There was a leukocytosis of 19,900. The urine was normal. A diagnosis of acute appendicitis could not be ruled out and operation was advised.

Operation.—The appendicectomy was performed through a McBurney's incision. The appendix showed no evidence of infection. There were several enlarged lymph nodes in the mesentery of the ileocecal region.

Microscopic Examination of the appendix revealed no evidence of infection. The patient had a very mild convalescence and was discharged 12 days after operation.

Case 5.—No. 88144. M. S., female, age nine, had been hospitalized for treatment of a congenital dislocation of the hip. She had been put in skeletal traction by means of a Steinman pin when her first symptoms occurred. The Steinman pin had been inserted November 2, 1934, under nitrous oxide anesthesia. She had had a mild convalescence, during which her rectal temperature ranged from 37.3° to 37.8° C., then dropped to 37.5° C. On the following evening it again rose to 38.4° C., and the patient became flushed and had generalized abdominal pain that centered about the umbilicus. She vomited twice and refused her supper. There was some tenderness in the right lower quadrant and the abdomen was somewhat distended. The throat was slightly reddened, and there was a slight amount of inflammation about the Steinman pin. The heart and lungs were normal; pulse 140; white blood count 26,750. On the following morning the white blood count was 18,000; the urine was negative except for many white blood cells. The urine examined was a voided specimen and the cells were thought to be from a vaginal dis-

charge. The child had a definite, atypical rash. The abdominal rigidity became greater on the right side and the tenderness extended from the right lower quadrant upwards and laterally to the kidney region. The white blood count was 22,150. A catheterized specimen of urine revealed clumps of renal epithelium cells and some white blood cells. The differential diagnosis apparently lay between an acute appendicitis, infection from the Steinman pin, and a right pyelitis. The severity of the symptoms and absence of definite diagnostic features made exploratory celiotomy advisable.

Operation.—A right midrectus incision was made under ethylene anesthesia. No free fluid was found in the abdomen. The appendix presented some fibrous adhesions as evidence of an old inflammation but otherwise was perfectly normal. It was removed. The right kidney region and the pelvis revealed nothing abnormal. The mesentery of the small intestine presented numerous enlarged lymph nodes. The wound was closed without drainage. Following operation the patient's temperature and pulse returned to normal within three days. The abdomen became soft and there was no further complaint of abdominal distress.

Microscopic Examination of the appendix revealed it to be normal. The wound healed by primary intention and the patient had a shelf operation for congenital dislocation of the hip three weeks later.

Case 6.—No. 75822. F. G., male, age seven and one-half, was treated for tuberculosis of the left foot and kidney during the year previous to his present complaint. The left kidney had been removed. His present complaint was acute generalized abdominal pain with nausea and vomiting, of three or four days' duration. His pain then became localized in both lower quadrants.

Physical Examination showed right lower quadrant rigidity and some right rectus spasm. There was tenderness on deep pressure in both lower quadrants, with some rebound tenderness. Rectal examination revealed some tenderness and a mass in the right iliac fossa. One hour after admission the white blood count was 16,300 and the temperature 38.2° C. The urine showed three plus albumin, was loaded with pus and contained an occasional red blood cell. A white blood count three hours after admission was 18,300. Because of the mass in the right lower quadrant and the rigidity at that point and the fairly typical history of acute appendicitis, it was decided to perform an exploratory celiotomy.

Operation.—The appendix was examined through a McBurney's incision. It appeared normal. The very small amount of free fluid in the abdomen was cultured. The mesentery of the terminal ileum revealed many enlarged lymph nodes 2 to 8 Mm. in diameter. This portion of the bowel appeared hyperemic, and a small nodule about 1 Mm. in diameter was present in its wall. There were two walnut-size lymph nodes in the mesenteric attachment of the cecum. A small lymph node in the mesentery was removed for examination. The appendix was removed and the wound was closed. The patient made an uneventful convalescence and the wound healed by primary intention.

Microscopic Examination of the appendix showed it to be normal. The lymph nodes showed only hyperplasia. Cultures of the fluid and lymph nodes were negative. About three months later the patient developed signs of tuberculous meningitis and died. He had had no abdominal complaints after operation. No autopsy was obtained.

Case 7.—No. 115483. S. F., female, age 13, was admitted to the hospital with a complaint of recurrent attacks of sharp, shooting pain in the right lower quadrant of two years' duration. She stated that the pain in the right lower quadrant in the first attack two years before admission was severe enough to cause her to lie down. The pain lasted two or three hours. Since that time she has had an attack about once a month, each attack persisting for almost a week. The pain was somewhat "shooting" in character and mostly in the right lower quadrant. It came on mostly at night and would last two or three hours. The remainder of the attack was characterized by a dull ache and tenderness in the right lower quadrant. The attacks were often accompanied by fever and occasionally by vomiting. Five months before admission she was seen during an attack

and a diagnosis of recurrent, subacute appendicitis was made. She was put on a liquid diet at that time. Two weeks before admission, the present attack came on, which has persisted. This episode has persisted longer than any previous one. She believed she had had some fever but there had been no vomiting. Some foods aggravate the pain. She had remained in bed most of the two weeks before admission. Her bowels had been regular.

Physical Examination.—The patient appeared very well nourished and did not look ill. The general examination was normal. Blood pressure 117/70; pulse 100. The heart and lungs were normal. An examination of the abdomen revealed tenderness in the right lower quadrant, localized to McBurney's point. No muscular spasm or palpable masses could be detected. The white blood count was 9,000; temperature 37.9° C.; urine normal.

It was thought best to observe the patient. No change was noted after 48 hours. Operation was performed, because of the frequency of the attacks and because of the parent's request.

Operation.—The appendix was examined through a muscle-splitting incision, and found to be somewhat hyperemic, but not acutely inflamed. The lymph nodes in the mesentery of the distal ileum were as large as 1 cm. in diameter. One was excised for study. The appendix was removed and the wound closed without drainage.

Microscopic Examination of the appendix revealed it to be normal. The lymph node was hyperplastic. Cultures of a portion of the lymph node gave no growth. The patient had an uneventful convalescence and was discharged a week later. She has had no recurrence of symptoms during the two months which have elapsed since operation.

Case 8.—No. 127288. B. S., female, white, age ten, was first seen May 1, 1935, complaining of abdominal pain of two days' duration. The patient had remained at home from school because of the abdominal pain on the right side accompanied by nausea and vomiting. This continued throughout most of the night. Her bowels had been normal but she was given an enema without results. This was then followed by castor oil. On the day of admission the pain had persisted, being located over the right side of the abdomen. The past history revealed that the patient had had ten attacks during the past seven or eight months, each consisting of cramping abdominal pain in the right side of the abdomen, usually lasting for several hours. She was frequently nauseated but did not vomit. On one occasion the attack was diagnosed as acute appendicitis but operation was not advised. The remainder of the past history was irrelevant.

Physical Examination.—The patient's face was flushed. Temperature 99.4° F. The tonsils were not large but the mucous membrane was reddened. The heart and lungs were normal; pulse 120. The abdomen presented tenderness over the entire right side and along the left costal margin. There was no rigidity. Rectal examination was normal. Urine negative. White blood cells 9,300; hemoglobin 81 per cent; and red blood cells 4,070,000. A diagnosis of acute appendicitis was considered and operation advised.

Operation.—On opening the abdomen the appendix was found to be grossly normal. Many enlarged lymph nodes were present in the mesentery of the small intestine. There was no free fluid. The appendix was removed and the abdomen closed without drainage. Microscopic examination of the appendix revealed no abnormalities. The patient had an uneventful convalescence. The pulse and temperature returned to normal and the patient was discharged one week after admission.

CASE HISTORIES OF FIVE PATIENTS IN WHOM A PRESUMPTIVE DIAGNOSIS OF MESENTERIC ADENITIS WAS MADE, BUT WHO WERE NOT OPERATED UPON

Case 9.—No. 106319. L. H., female, age ten, was seen August 11, 1934, complaining of a sharp pain in the right side, of 24 hours' duration. The mother stated that for two or three days the child had had a poor appetite and had not been playing. During the evening before admission there had been a gradual onset of right lower quadrant pain which later shifted to below the ribs. The child slept well that night but the following morning the pain was quite severe and had remained so all day. There had been no

nausea or vomiting and the bowels had been normal. There had been no urinary complaints. There was a history of two other attacks of less severity, the first two years and the second three months previously. The past history otherwise was irrelevant.

Physical Examination revealed a well developed girl who seemed acutely ill. She lay on her right side to ease her pain. The findings on general examination were normal except for large, cryptic tonsils. Blood pressure 105/55; pulse 90. Examination of the abdomen disclosed slight muscular rigidity, tenderness both in the upper and lower right quadrants, somewhat more pronounced in the lower. Temperature 38.9° C. White blood cells 12,500; 95 per cent hemoglobin; red blood cells 4,620,000. The urine findings were normal. Acute appendicitis was suspected but because of the lack of localized findings it was thought best to defer operation temporarily and to observe the patient. Two subsequent white counts showed little change. However, on the following day two white counts were 6,500 and 6,800, and the temperature had returned to normal. The patient was discharged a day later. She was seen in the Clinic two days after discharge, at which time there were no complaints and no abnormal findings. She was asked to return if further symptoms developed but has not done so.

Case 10.—No. 99438. P. D., male, age five, was seen first August 12, 1934, for a complaint of abdominal pain of three days' duration. On August 10 he awoke and told his mother that he had felt like vomiting before he went to sleep. During that day and on the previous day, he had had three or four watery, light brown stools and had complained of pain in the abdomen. The mother thought the child's pain was worse on the right side. On the following morning he was quite cross and irritable and complained of a frontal headache and of abdominal pain, especially on coughing. He continued to eat his meals regularly and had no stools that day. He went to bed at the usual time but awoke two and one-half hours later screaming with pain and doubled up in bed. There was no history of the patient's having had anything out of the ordinary to eat.

Physical Examination revealed a well developed boy whose abdomen was held quite rigid. There was generalized tenderness but more so on the right side than on the left. The findings on rectal examination were normal. White blood cells 11,600. The urine was normal. Because of the lack of localized findings a definite diagnosis was not made and observation was advised. A white cell count taken one hour later was 13,700 and on the following morning it was 13,600, and about the same six hours later. However, by this time there was very little pain and although there was some tenderness over the entire right abdomen, it was not marked and there was only a very slight degree of right rectus rigidity. Temperature on admission was 38.6° C.; 24 hours later it was normal and remained so thereafter. The white count remained as high as 12,000 although the findings and symptoms had completely subsided. The patient did not keep a return appointment in the Out-Patient Clinic.

Case 11.—No. 55205. D. G. S., female, age 11, was admitted February 12, 1932, with a complaint of intermittent abdominal pain confined usually to the right side. Six weeks before admission the patient first noticed intermittent abdominal pain. One day it was accompanied by vomiting and diarrhea and was so severe that she was confined to bed. Temperature 38.9° C. Since then she had had an average of two attacks a week characterized by sudden onset of abdominal pain which would "double her up." These attacks were not accompanied by vomiting or other symptoms and have not prevented attendance at school.

Physical Examination revealed a well developed, well nourished girl. She appeared somewhat pale and her pharynx was injected. The general examination was normal, except for some coarse râles in the right apex. Her abdomen was somewhat tender in both lower quadrants. Temperature 37.6° C. Examination of the blood and urine revealed no abnormal findings. The Mantoux test with 1 : 100 tuberculin was negative. Roentgenologic examination of the chest and of the genito-urinary tract revealed no abnormalities. The diagnosis at this time was slight upper respiratory tract infection and mesenteric lymphadenitis. She was seen again February 27, 1932, when a small mass was palpated

in the left lower quadrant. There were no complaints at that time. She was seen at intervals of four to eight weeks during the next two and one-half years. The mass in the left lower quadrant remained about the same size and was fairly freely movable until January, 1934, when it appeared to be getting smaller. However, it was still palpable when the patient was last seen in June, 1934. It was not tender and quite freely movable and was thought to be a mesenteric lymph node. There was no recurrence of abdominal pain or of vomiting.

Case 12.—No. 74086. R. M. D., female, age six, was admitted April 27, 1935, complaining of diarrhea and vomiting of two days' duration. Two days before admission the patient complained of a distended abdomen. The following day she had not felt well but ate her meals. About ten o'clock, the night before admission, she began vomiting and had several liquid stools; there was low abdominal pain of a cramping nature. The pain was localized first around the region of the umbilicus and later in both lower quadrants. There was no respiratory infection. The abdominal pain continued up to the time of admission.

Physical Examination.—The patient did not appear acutely ill. The abdomen was generally rigid. She complained very little of pain or tenderness on examination. Rectal examination was normal and on the insertion of a rectal tube, a liquid stool was obtained. General examination was otherwise quite normal. White blood cells 18,000; urine normal; temperature 37.7° C.

It was decided to observe the patient over night. Saline and glucose were given subcutaneously. On the following day, the white count was about the same and the urine was normal. Her rectal temperature remained below 38° C. during her entire stay in the hospital, which was five days. Four days after admission, the white count was 14,000; the abdomen became soft and there was no more vomiting or diarrhea during her entire stay. The stools were negative for *Endamœba histolytica.* Roentgenograms of the chest were normal.

Case 13.—No. 62254. R. C., male, white, age seven, was admitted January 29, 1935, complaining of abdominal pain of six months' duration. The patient had been quite well until September, 1933, when he had an attack of severe vomiting and abdominal pain and a fever as high as 105° F.

He was seen at the Cook County Hospital, where little rigidity or tenderness was found on examination, and he was allowed to go home. He was readmitted October 3, 1933, and a normal appendix removed. The patient stated that several days after the operation the wound broke open and drained, off and on, for several weeks. He was well until the following August, 1934, when he had an attack of diarrhea and pain in the abdomen which caused him to "double up." The pain was located around his waist and he was treated for colitis. The attack occurred every few weeks thereafter, until his present trouble which began a few days before admission and was very similar in character to those previously suffered.

The past history revealed that in March of 1933, six months before the first described attack, the patient was seen at a Pediatric Clinic having symptoms of bronchitis and pain in the abdomen. A diagnosis of mesenteric lymphadenitis was made at that time. On questioning the mother, she stated that the recurring attacks of abdominal pain were definitely related to recurring upper respiratory tract infections.

Physical Examination.—The patient did not look acutely ill. There was evidence of a subsiding pharyngitis with enlarged cervical lymph nodes. The heart and lungs were normal. The abdomen was full and was held voluntarily tense all over, but was not tender. There were no masses. An operative scar was present in the right lower quadrant. Rectal examination was negative. Urine normal. White blood cells 8,500; hemoglobin 85 per cent; red blood cells 4,800,000. A tuberculin test of 1 to 1,000 dilution was negative, and examination of the stool was negative for parasites. Roentgenologic examination of the G. I. tract was normal and showed no calcification of the mesenteric nodes. A diagnosis of subsiding upper respiratory infection and subacute mesenteric

lymphadenitis was made. The patient was put on a controlled medical regimen and discharged one week after admission. He was followed in the Out-Patient Clinic, and when seen three months later had been having no further trouble.

TABLE I

RÉSUMÉ OF 13 CASES OF NONTUBERCULOUS LYMPHADENITIS

	Number of Cases
Incidence	
Season	
Spring	4
Summer	1
Autumn	5
Winter	3
Repeated attacks	9
Associated with upper respiratory infections	8
Operated	8
Symptoms	
Nausea	10
Vomiting	9
Diarrhea	5
Colic	10
Pain	
Epigastric	1
Umbilical	3
R. L. Q.	2
Right side	4
Generalized	6
Signs	
Temperature	
37.0° to 38.5° C.	9
38.5° C.	4
Tenderness	
Epigastric	1
Umbilical	0
R. L. Q.	3
Right side	3
Generalized	3
Rigidity (including resistance)	
R. L. Q.	1
Right side	3
Generalized	5
Findings	
W. B. C.	
8,000 to 15,000	5
15,000	8
Culture of lymph node	2
Positive for hemolytic streptococcus	1
Guinea-pig inoculation	2
Positive for tubercle bacilli	0

Etiology.—The factors which contribute to the production of nontuberculous lymphadenitis remain obscure. Mead[6] has discussed the etiologic

theories on this subject advanced by various authors. When the disease first became a definite clinical entity, all cases were thought to be tuberculous. This misconception was corrected when guinea-pigs injected with material from nodes removed at operation remained free of tuberculosis. The Mantoux test, also, did much to differentiate the two types preoperatively and to establish the nontuberculous variety as a definite group of cases of different etiology. Recent reports reveal the nontuberculous type to be much more common than the tuberculous.

Of all possible etiologic factors of the nontuberculous type, the appendix has probably received most consideration. Although most cases have become symptom free following appendicectomy, this is not proof that the appendix was at fault. That many cases become symptom free on controlled medical management, and further that enlarged mesenteric nodes rarely are present in cases of uncomplicated acute appendicitis, are convincing evidence that the appendix is not the cause of the condition. Further evidence against this theory is the routine finding of an appendix which reveals no gross or microscopic pathologic change and the persistence of symptoms in a few cases following appendicectomy.

The colicky, intermittent character of the abdominal pain, with diarrhea in some cases, and dehydration, suggests the intestine as a likely etiologic factor. However, at operation the bowel appears normal on gross inspection. That the condition may be only a local manifestation of a generalized condition must be strongly considered. Nine of our 12 cases of the nontuberculous type occurred in the spring or autumn, and eight were definitely associated with an upper respiratory infection. In one case the abdominal symptoms recurred with each flare-up of his upper respiratory condition; this after a normal appendix had been removed, elsewhere, two years previously. Both conditions subsided completely on controlled medical management. In one case hemolytic Streptococci were isolated from a mesenteric lymph node. This finding is in accord with that of Goldberg,[4] who cultured the Streptococci from the throat and from the nodes in a series of cases.

Clinical Manifestations.—Two of the most outstanding features of this condition are the colicky nature of the abdominal distress and the marked tendency for recurrence of attacks. This has been reported repeatedly by other authors and was observed in most of our cases. Not infrequently there is a history of a recent upper respiratory infection, and it is not unusual to find either an active condition present or remnants of a previously existing one. Although some authors have reported the greatest age incidence to be between 20 and 30 years (Freeman[3]), most cases are perhaps younger than this. All of the cases herein reported were less than 14 years of age.

The attacks usually come on quite suddenly and the patient is "doubled up" with cramping pain in the abdomen, which lasts from a few minutes to one or two hours. In half of our cases this pain remained generalized and in only two was it localized in the right lower quadrant. The attacks were

accompanied by nausea and vomiting in most of the cases and five cases had an associated diarrhea.

Examination revealed tenderness localized to the right lower quadrant in only three cases. The others presented tenderness over both sides of the abdomen or over one entire side. Right rectus rigidity was present in only four cases. The others presented a generalized resistance or none at all.

Another striking feature is the frequency of an elevated white cell count out of all proportion to the patient's temperature. This seemed to be more obvious in the earlier attacks. Several of our cases had a leukocytosis of 20,000 or above, and one had a white cell count of 41,000; only four had a temperature over 38.5° C. Abdominal masses usually are not palpable in the nontuberculous variety.

Diagnosis.—A definite diagnosis of nontuberculous lymphadenitis in most cases is very difficult to make before operation. Its clinical picture is so variable that McFadden[5] says: "There is hardly an abdominal condition for which laparotomy is commonly performed that may not be simulated by diseased mesenteric glands." It is almost invariably confused with acute appendicitis and must also be differentiated from pyelonephritis, intussusception, and Meckel's diverticulitis.

The history of many previous attacks and the colicky nature of the abdominal pain together with the lack, in many cases, of localizing signs or symptoms are helpful in arriving at a definite diagnosis. When possible, observation of the patient for a few hours following admission may be very helpful. Nine of our 13 cases were operated upon. One of these was of the tuberculous variety and one had had an appendicectomy elsewhere about a year before admission.

Pathology.—At operation all of our cases presented practically the same picture. There was usually little or no free fluid in the peritoneal cavity and, if present, was sterile on culture. The appendix appeared normal or slightly hyperemic. The peritoneum of the mesentery and the bowel wall appeared normal. The mesenteric lymph nodes at the ileocecal junction and extending upward on the ileum were enlarged from 0.5 to 1.5 cm. in diameter and were somewhat flattened. The greatest collection of enlarged nodes was at the ileocecal junction. They were discrete and pinkish in color and on removal presented a granular appearance. Other authors have reported finding enlarged nodes in various parts of the mesentery, some showing necrosis, caseation, or calcification (Mead[6]). Exploration was limited in our operated cases and autopsy was denied in one fatality. Thus the findings are limited to a local area.

Microscopic sections revealed only lymphoid hyperplasia.

A *Streptococcus hemolyticus* was cultured from a removed node in one of two cases and guinea-pig inoculations were negative in the two cases in which this was done.

The one case of tuberculous mesenteric lymphadenitis presented several very large (2.0 cm.) nodes matted together near the ileocecal junction.

There were many smaller ones in the mesentery of the ileum extending above this region.

Treatment and Prognosis.—In most cases where the diagnosis is uncertain, exploratory celiotomy is indicated. In some cases it is possible to observe the patient for several hours before a definite type of treatment is decided upon. Restoration of fluid and mineral balance and limitation of intake by mouth to a minimum may be carried out while waiting to decide if operation is advisable.

A rapid subsidence of abdominal symptoms and pathologic findings is very helpful in determining the true nature of the condition. This period of observation need not be longer than two or three hours, and if the symptoms and findings make delay seem hazardous, exploration should be carried out at once. The danger accompanying exploratory celiotomy and appendicectomy is little compared to that associated with complications arising from a mistaken diagnosis.

When exploration was undertaken, removal of the appendix was the routine. Lymph nodes were removed only for diagnosis.

Except for the one fatality all cases of the nontuberculous variety, whether operated upon or not, have made complete recoveries on a controlled medical, dietary and hygienic management. All authors have stressed these as important measures in the treatment of the disease. There is little evidence to support the opinion that appendicectomy is necessary to obtain a permanent cure.

The one case of the tuberculous type died several months postoperatively from tuberculous meningitis.

SUMMARY AND CONCLUSIONS

(1) Twelve cases of nontuberculous and one of tuberculous mesenteric lymphadenitis have been reported and discussed.

(2) Although the appendix and small intestine have received the major consideration as etiologic factors in the production of this condition, it is not unlikely that enlarged mesenteric lymph nodes are only a local manifestation of a generalized condition having its origin in an upper respiratory tract infection.

(3) The colicky nature of the abdominal distress, the multiplicity of attacks with relatively short duration and frequent association with an upper respiratory tract infection are outstanding characteristics of the disease.

(4) The disease is chiefly one of children, all of our cases being under 14 years of age. The average age reported by most authors is below 20 years.

(5) Nontuberculous lymphadenitis must be differentiated from acute appendicitis, pyelonephritis, Meckel's diverticulitis, and intussusception.

(6) The diagnosis usually is made at operation. However, the colicky nature of the pain and lack of localized symptoms or findings in cases which may be observed for a few hours may lead to a correct diagnosis without

operation. This is especially true where there is a rapid subsidence of symptoms and findings; a course which is often observed in this condition.

(7) Simple lymph node hyperplasia of the mesenteric nodes in the region of the ileocecal junction and, to a lesser extent, in the mesentery of the lower ileum, is the usual pathologic finding. Occasionally pyogenic organisms may be cultured from these nodes.

(8) Exploratory celiotomy is the treatment of choice, especially where a definite diagnosis cannot be made. The appendix is removed and the wound is closed without drainage.

(9) The prognosis for a permanent cure is very good. Most cases remain symptom free following appendicectomy. This is also the case where controlled medical management and antituberculosis measures are observed in nonoperated cases.

(10) Only one case of tuberculous lymphadenitis has been observed as compared to 12 nontuberculous cases; thus showing the relative incidence of the two. This conforms favorably with the statistics of other authors.

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THE PATHOGENESIS OF NECROSIS OF THE CEREBRAL GRAY MATTER FOLLOWING NITROUS OXIDE ANESTHESIA *

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DURING the past five years, the writer has had the opportunity of studying the clinical manifestations and, in the fatal cases, the pathologic alterations in a number of patients who presented untoward symptoms, indicative of cerebral damage, after nitrous oxide anesthesia. A résumé of the 13 cases studied, nine of which came to autopsy, have been reported elsewhere.[1]

As a rule, these patients developed respiratory or cardiorespiratory failure while under nitrous oxide-oxygen anesthesia and failed to regain consciousness when the anesthetic was withdrawn. During the survival period the patients remained in coma, frequently had convulsions and most of them died in a state of hyperthermia after an interval of one and one-half to 26 days. A few individuals recovered, some with a residual lenticular syndrome,[2] others with a permanent psychosis,[3] while some fortunately recovered completely. In the fatal cases, the cerebral cortex and the lenticular nuclei presented areas of necrosis which at times became confluent and, if the survival period was sufficiently long, resulted in astrovascular scars. Because of their histologic characteristics, these lesions were believed to be the result of asphyxia, a conclusion which was supported by Dr. Yandell Henderson.

In a recent study by Lowenberg, Waggoner and Zbinden,[4] four similar cases were reported, pathologic examinations of the brain being made in the three fatal cases. The cerebral cortex and basal ganglia presented characteristic areas of necrosis. As a result of their observations, these investigators concluded that the cerebral damage was due to the toxic action of nitrous oxide.

Thus both of the two possible causes have been held to be responsible for the injury to the cerebral gray matter. The writer has taken occasion to review his material to see whether the morphologic changes found gave support to either of these two theories. This review was made with a realization of the difficulty of attempting to explain abnormal physiology on the basis of structural alterations. A final conclusion can be reached only after the problem has been attacked from the physiologic and pharmacologic aspects as well.

Mode of Action of Nitrous Oxide.—It is necessary to review briefly the mode of action of nitrous oxide in anesthesia, to determine whether its action

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is due primarily to some specific narcotic effect or to an associated or resultant asphyxia. From a physiologic standpoint, nitrous oxide does not behave entirely like a pure asphyxial gas: (a) The pulse is not so slow (the cardio-inhibitory center is less capable of activity); (b) convulsive movements are less marked (due to depression of the motor nerve cells); and (c) respiration ceases earlier (due to a specific depressant action on a respiratory center). This specific depressant action of the gas is noted when it is used even in dilute solution with oxygen, distinct narcotic effects being produced in proportions of one to four. It has also been shown that this narcotic action disappears immediately when nitrogen, which is an inert gas, is substituted for nitrous oxide, under which circumstances the patient promptly recovers. These observations indicate, therefore, that nitrous oxide does exert a distinct narcotic effect *per se*.

How much of the effect of nitrous oxide is due to asphyxia? The early investigators, Bert[5] and Martin[6] were of the opinion that death was due to lack of oxygen rather than to any depressant effect of nitrous oxide on the respiratory center. From a purely clinical standpoint, Hewitt[7] believed that the obstructive stertor, convulsive movements and cyanosis occasionally seen with nitrous oxide anesthesia were of anoxemic origin and were not essential to anesthesia. As a result of their experimental investigations as to the amounts of oxygen, carbon dioxide and nitrous oxide concentration in the blood of dogs under nitrous oxide anesthesia, Leake and Hertzmann[8] concluded that surgical anesthesia was invariably accompanied by some degree of anoxemia. Greene[9] and his collaborators reached the same conclusion and found that a high concentration of the gas was necessary for surgical anesthesia in experimental animals. Their work made it seem evident that the depth of anesthesia depended not as much on the concentration of nitrous oxide in the gas breathed as it did on the variations of the percentage of oxygen.

Brown, Lucas and Henderson[10] state that "patients anesthetized with nitrous oxide will always suffer from a severe degree of anoxemia and that this must impose a limitation upon its use and increase its danger." As a result of experimental work and of clinical observation,[11] one must, therefore, conclude that surgical anesthesia with nitrous oxide is accompanied by advanced degrees of anoxemia and, therefore, that anoxemia must be given serious consideration as a possible cause of untoward neurologic manifestations.

The Toxicology of Nitrous Oxide.—The undesirable manifestations of any anesthetic agent may be the result of its inherent toxic properties or the production of undesirable physiologic states which it creates or due to secondary complications which it encourages. The untoward effects of nitrous oxide may well be considered on this basis. Experimentally, it seems fairly clear that the actual narcotic action of nitrous oxide is of little moment insofar as its fatal effects are concerned. This is shown by the fact that animals die in about the same length of time after inhalation of nitrous oxide as with an inert gas

such as hydrogen.[12] Its anoxemic or asphyxial effect must, therefore, be given consideration as the cause of untoward symptoms.

Anoxemia and Nitrous Oxide Anesthesia.—There are three types of anoxemia depending upon the factor producing it (Barcroft). The *anemic type* is due to deficient amounts of hemoglobin which makes it impossible to carry a normal amount of oxygen, even though the oxygen tension in the blood is normal. The *stagnant type* of anoxemia results from a slowing of the circulation, as following cardiac failure. The oxygen content and the oxygen tension are normal under these circumstances but the amount of oxygen actually supplied to the tissues in a given interval of time is reduced. The *anoxic type*, which is the most serious of the three, is a result of lowering of oxygen tension in the blood so there is actually less oxygen in solution. This usually results in a coincident lowering of the carbon dioxide tension which further decreases the rate of dissociation of hemoglobin so that the oxygen present is not so readily available to the tissues. In nitrous oxide anesthesia we are dealing with anoxic anoxemia resulting in a reduction in the actual amount of oxygen available in the blood. This is true even when pure oxygen is given under pressure with the gas. The oxygen tension in the blood is lowered and, therefore, the amount of oxygen available to the cerebral cells is reduced. This anoxic type of anoxemia may be accentuated by mechanical difficulties in the apparatus and obstruction of the respiratory passages.

The untoward symptoms of nitrous oxide anesthesia may be mild or severe. The mild transient symptoms are cyanosis, respiration, irregularities and muscular twitchings or grosser, uncontrolled muscular movements or even convulsions. At times stupor or mental cloudiness may persist for a variable interval after withdrawal of the anesthetic. Restlessness, irrationality, hysterical outbursts or most prolonged psychoses have also been described. Among the more serious eventualities are more or less severe and persistent neurologic manifestations of psychic, motor or sensory character. Persistent manifestations indicative of injury to the lenticular nucleus may be present in some cases. In others the resultant psychosis may be so marked as to require hospitalization. Such patients may succumb after an interval of days, weeks or even months after anesthesia. It is in this group of cases that cerebral damage can be demonstrated postmortem. In another group of cases, sudden death occurs under the anesthetic. Excluding those cases with an advanced surgical lesion, the immediately fatal issue has been attributed to persistent thymus, idiosyncrasy, pressure on the carotid sinus or to direct depression of the respiratory center by the gas.

In a recent report Lowenberg, Waggoner and Zbinden[4] analyzed a series of four patients who presented serious residual manifestations, and discussed the mechanism of cerebral change. They believe that: (*a*) Asphyxia following nitrous oxide anesthesia may be produced by lowering the oxygen content of the blood; or (*b*) That it may be due to collapse of the cerebral capillaries. In discussing the first of these two propositions, they cite a number of German authors who advocate the conception that asphyxia is due to lowered oxygen content of the blood. These observers have inadvertently fallen into the error of assuming that the absence of cyanosis is indicative of the absence of anoxemia. That the two symptoms are not necessarily synonymous has been amply demonstrated.[13] In these cases, because cyanosis does not occur, it cannot be con-

cluded that anoxemia is not present. Furthermore, the very important influence of respiratory and cardiac failure as the means of prolonging and intensifying the local cerebral asphyxia has been underestimated.

Discussing the second possible cause of cerebral damage, namely, as a toxic effect, these authors point out the results of the experiments of Gianotti and Vannotti,[14] who studied the effects of nitrous oxide on the cerebral capillaries of experimental animals (rabbits). It was found that while anesthesia was being inducted, there was a contraction of the entire capillary system which was followed by dilatation as soon as anesthesia was established. In other words, cerebral congestion followed nitrous oxide anesthesia. This observation seems to be in accord with the findings at autopsy in individuals who die within a certain interval following nitrous oxide anesthesia. In these cases the cerebrum is found to be markedly congested, both grossly and microscopically. This conclusion is also supported by the fact that the brain bulges through any operative defect in the skull in patients under nitrous oxide anesthesia, a situation which is relieved by the administration of oxygen. This congestion is naturally followed by a slowing of the blood current. Gianotti and Vannotti did not find a collapse of the smaller blood vessels nor did a complete stasis result. This temporary slowing of the blood current was immediately counteracted by administration of oxygen. They emphasized the difference between nitrous oxide-oxygen anesthesia and nitrogen-oxygen asphyxia in which there is a collapse of the blood vessels and spaces. Lowenberg, Waggoner and Zbinden conclude therefrom that, since there is no collapse of blood vessels, the cortical damage cannot be due to asphyxia but to some toxic action of nitrous oxide.

Significance of Cerebral Changes Following Nitrous Oxide Anesthesia.—A survey of the changes in the brain following delayed exitus after nitrous oxide anesthesia together with their possible significance is considered. In cases with shorter survival periods, the earliest evidence of damage is found about the pericellular spaces of the pyramidal cells of the cerebral cortex. The nerve cells in the area affected undergo acute alterations and the surrounding interstitial tissue are found to be vacuolated (Fig. 1). Involving only a small group of cells, these areas may be so limited in extent as to escape the notice of a casual observer. Only when these areas attain sufficient size to produce a pale area (*Herde*) in the cortex on stained section do they become evident with lower power magnification (Fig. 2).

Is this finding indicative of a narcotic or an asphyxial action? It is known that narcotics exert a specific effect on the nerve cells resulting ultimately in morphologic alterations in these cells. In severe cases of poisoning, a second effect may be evident, one which is due to alteration of the endothelium of the blood vessels, permitting an escape of red cells into the perivascular space (ring hemorrhage). There is, however, no direct action of narcotics on nerve fibers or interstitial elements. On the other hand, any or all structures seriously affected by asphyxia undergo morphologic change. Insofar as the brain is concerned, infarctions consequent to occlusion of a local blood vessel are classical examples of asphyxia. On microscopic examination of an infarcted area, we find that both the nerve cells and interstitial structures are definitely injured.

Judging from what is known about the effect of narcotics on one hand and of vascular occlusion on the other, we must, therefore, conclude that these local minor alterations in the cortex are more likely the result of asphyxia rather than of narcosis.

Attention should now be given to the next most serious lesion of the brain, namely, patchy or spotty necrosis (Fig. 3). Are these lesions an evidence of narcosis or of asphyxia? Gildea and Cobb[15] have shown that similar areas are to be found in the cortex after temporary ligation of the large vessels supplying the brain. These experimental results are reproduced remarkably well in the patients who survive for a sufficient interval following nitrous oxide anesthesia.

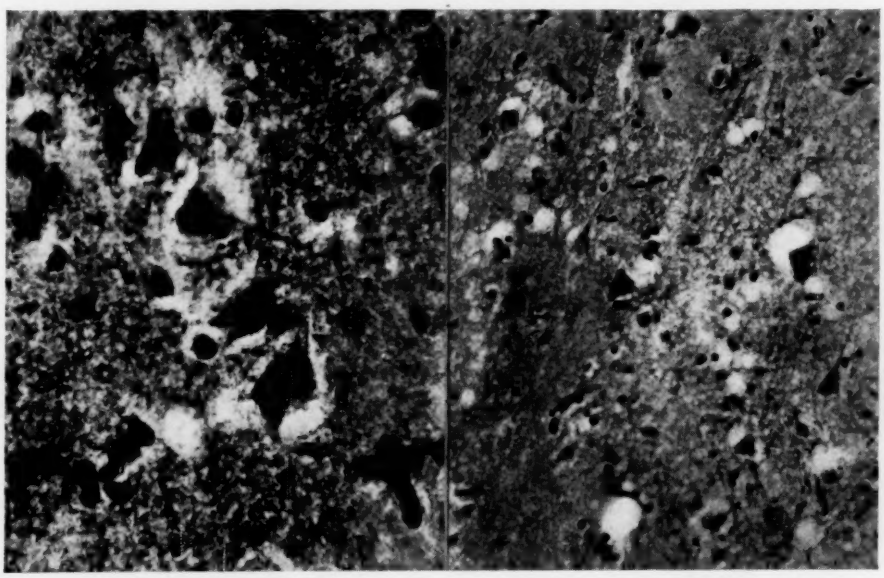

FIG. 1.—Early changes in the cerebral cortex. Enlarged pericellular spaces and vacuolization of the surrounding interstitial tissue and nerve cells (H. & E. × 620).

FIG. 2.—Area of destruction showing enlarged pericellular spaces and vacuolization of the interstitial tissues. The margin of the area is clearly defined (H. & E. × 250).

This fact would strongly indicate that this lesion, found characteristically after nitrous oxide anesthesia, is essentially an effect of asphyxia.

By carrying the analogy one step further, it is found that these areas of destruction or spotty necrosis tend to occur in the vicinity of blood vessels (Fig. 4). The blood vessels in these areas of devastation are often found to be dilated as compared to those in undamaged areas. This dilatation of the blood vessels which is probably an effect of nitrous oxide, is followed by a slowing of the blood stream. The amount of oxygen reaching any particular part is, therefore, definitely reduced (stagnation). This seems to be a conclusive argument that focal necrosis is not a narcotic effect, for stagnation would mean that *less* nitrous oxide would reach the affected area. As final proof against this conception, is the fact that narcotics are not known to produce focal areas of necrosis in the cerebral gray matter.

Turning now from these relatively minor alterations, we examine the more serious and profound lesions sometimes observed in these cases. In patients

FIG. 3.—Isolated areas of necrosis in the cerebral cortex. Note relationship of one area to the local blood vessel (Cajal reduced silver preparation × 22).

FIG. 4.—Areas of necrosis about the dilated blood vessels in the lenticular nucleus (H. & E. × 50).

FIG. 5.—Fusion of areas of destruction in a more advanced lesion (Cajal's reduced silver preparation × 22).

who survive for a sufficiently long interval following cardiorespiratory failure under nitrous oxide anesthesia, more or less complete localized destruction of the cortex may occur (Case 9 of author's series with survival for 26 days) (Fig. 6). The lenticular nucleus, particularly the globus pallidus, are also seriously damaged. Does the extent and distribution of these lesions indicate whether this effect is a narcotic or an asphyxial one? In the first place, there is no narcotic which is known to produce so extensive a lesion. For example,

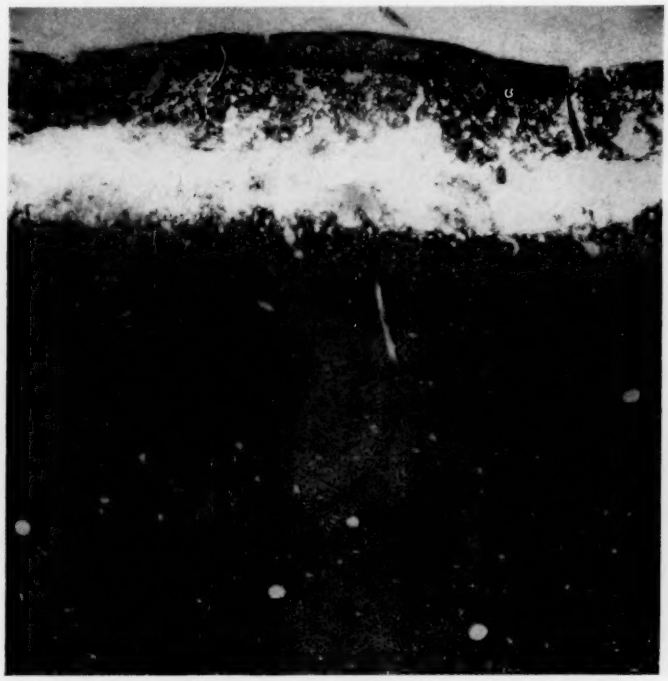

FIG. 6.—Subtotal destruction of the visual cortex. Survival period 26 days (Cajal's reduced silver preparation × 22).

severe morphine poisoning will cause injury to individual nerve cells as well as result in petechial hemorrhages throughout the brain, particularly in the white matter. Following nitrous oxide asphyxia, petechial hemorrhages are the exception rather than the rule. Furthermore, destruction of the lenticular nuclei is a more or less classic pathologic manifestation of asphyxia from almost any cause, such as carbon monoxide poisoning which also provokes an anoxic anoxemia.

Finally, the irregular distribution of these areas of destruction and the variability of severity of the lesion in various portions of the cortex is not typical of narcotic poisoning in which the effects are more or less diffuse and uniform. There is no known narcotic, for example, which has a predilection for the visual area of the cortex, while this is not uncommon following nitrous oxide anesthesia. One must conclude again that on the basis of the severity and distribution of the lesions, the pathologic situation is more characteristic of asphyxia than of narcosis.

Discussion.—Space does not permit a complete consideration of the possible predisposing factors of the clinical manifestations present in these cases. These have been quite adequately discussed in the author's original study.[1, 2] A few clinical points will serve to emphasize certain facts which are pertinent.

In the first place, it is necessary to reëmphasize the important physiologic fact that the absence of cyanosis does not mean an absence of asphyxia, since the one may occur without the other. Furthermore, it is quite obvious from the clinical behavior of these cases that the mechanism of asphyxia must be a variable one. In some instances, it is a prolonged and gradually accumulative effect. In most cases, however, it is abruptly precipitated by sudden respiratory and cardiac failure, possibly incident to a depressant action of the gas on the vital centers or at times to the development of some intercurrent lesion (such as pulmonary embolism). It is to these cases with prolonged respiratory and cardiac failure that particular attention must be given.

Without doubt, the major factor in the production of cerebral damage in these cases is not so much due to the presence of nitrous oxide itself, but rather to the failure of circulation in the brain during this interval of cardiac failure. The blood, already poor in available oxygen, soon becomes depleted of this gas, and since no additional oxygen is supplied owing to failure of circulation, the destructive effects of anoxemia take place almost immediately. It is doubtless during this interval that the cerebral damage occurs. The most severe effects are evident about vessels which happen to be dilated at the moment, as indicated by the perivascular location of the areas of necrosis. Stagnation is, therefore, probably the important factor which ultimately designates the location of the areas of necrosis. Cortical damage is due, therefore, not so much to the presence of nitrous oxide, which is probably diffused proportionately throughout the blood stream, as it is to the failure of respiration and circulation. The cortex does not get an adequate supply of oxygen and permanent damage results.

CONCLUSIONS

The peculiar damage to the cerebral cortex and lenticular nucleus occurring in most instances after serious cardiorespiratory failure under nitrous oxide anesthesia, is probably the result of asphyxia and is not due to toxic narcotic action of the gas. This seems to be evident by the fact that the earliest effect is observed in the interstitial tissue about the pericellular spaces as well as in the nerve cell itself, which is characteristic of asphyxia and not of narcosis. Furthermore, in the areas of patchy necrosis, the findings are those characteristic of oxygen want, similar to those produced experimentally by temporary ligation of the larger arteries of the brain. These areas of necrosis are not observed after poisoning with narcotics. The extent and distribution of areas of severe necrosis in patients with longer survival periods are likewise not characteristic of any known narcotic. Degeneration of the lenticular nucleus, on the other hand, is a common effect of asphyxia as seen in carbon monoxide poisoning.

These alterations are probably the result of an anoxic anoxemia, incident

to the lowered oxygen content and tension in the blood, an accompaniment of nitrous oxide anesthesia. When there is a sudden failure of respiration and cardiac action, the reduced amount of available oxygen is promptly used up and since no additional amount of oxygen is supplied, the brain suffers from the lethal effects of asphyxia. The localization of the areas of focal necrosis seems to be dependent upon dilatation of the vessel with consequent stagnation. Why all vessels are not coincidentally affected is not known.

One may conclude, therefore, that the cerebral damage is a direct result of asphyxia, even though at times the cessation of respiration and cardiac action may be due to a depressant (narcotic) action of the gas on the vital centers. In some of these cases, however, cardiorespiratory failure is due, not to the narcotic effect of the gas but rather to the development of some intercurrent lesion such as pulmonary embolism. In cases without cardiorespiratory failure, cerebral damage is probably the result of prolonged accumulative anoxemia of a dangerous degree, due to inherent predisposing factors in the patient (alcoholism), to defects in the apparatus, to impurities in the gas or perhaps at times to improper administration of the anesthetic.

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GENERALIZED LYMPHOGRANULOMA INGUINALE

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LYMPHOGRANULOMA inguinale was so named by Durand, Nicolas and Favre[17] in 1913, because they believed that its lesions were limited to the genitalia and inguinal lymph nodes. Today it is certain that that conception is incorrect, but it is equally certain that the complete picture of the disease is not yet known. It is even possible, as has been suggested by Coutts,[11, 12] Martin and Bacon,[35] and others, that there are really two diseases included in this classification, though, so far, there has been no proof of their contentions. The purpose of this paper is to emphasize some aspects of this disease which have received but little attention in the American literature.

The diagnosis of lymphogranuloma inguinale demands both its own laboratory and clinical confirmation and the exclusion of other diseases, which may in part account for the late recognition of it as an entity. Neither the clinical picture, the histology, nor the Frei test can be depended upon alone. Thus, although one form of "The Venereal Disease," described by John Hunter[27] in 1786, agrees completely with the usual inguinal form of lymphogranuloma inguinale, nevertheless, those cases may quite well have been either tuberculosis or syphilis. Throughout the nineteenth century there were reports of cases of "climatic bubo," many of which are now supposed to have been lymphogranuloma inguinale, but for which there is no proof (Chassaignac,[7] Trousseau,[50] Nelaton,[37] and Klotz[29]). Even after Nicolas and Favre's[17] detailed description, and after the development of methods for the laboratory identification of syphilis, tuberculosis, chancroid, Hodgkin's disease, granuloma inguinale, and plague, the diagnosis was still largely that most unsatisfactory sort, the diagnosis by exclusion, and it remained so until 1925, when Frei[19] published his diagnostic skin test. Likewise, the concept of the disease remained necessarily closely limited to the one originally outlined 12 years before.

Ravault and Rabeau,[43] in 1922, raised the question of possible generalization after noticing that some of their patients showed fever, malaise, and transient positive Wassermann reactions, but it was not until 1928 that Frei and Koeppel[20] demonstrated the extension of the disease outside of lymph nodes into the rectal wall and perineal soft tissues. Later in the same year Bergstrand[1] published the first conclusive case of widespread lymphoid involvement, confirmed by biopsies and repeated Frei tests.

Since then the story of lymphogranuloma inguinale has been one of ever widening concepts. In 1931, Buschke and Curth[4] reported a case of primary

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infection in the mouth, and, in 1932, Chevallier and Bernard[8] a purely cutaneous form of the disease. Luján and Rotter's[34] instances of contact infection in children appeared the same year, as did the first comprehensive article in the American literature on the disease as a whole, that of DeWolf and Van Cleve.[16] Frauchiger[18] reported his observations on polyarthritis in 1933. The next year Škorpil[48] reported a case of generalized lymphadenitis similar to our case, and Kitagawa[28] and Midana and Vercellino[36] published their observations on the changes in the central nervous system. The recognized extent of the disease was further widened in 1935 by Reichle and Connor,[45] who reported a case of death from suppurative arthritis of the hip and interstitial nephritis of lymphogranulomatous origin. The first report of death from a fulminating meningo-encephalitis was made by Rajam[42] in 1936, and primary granular conjunctivitis was also added to the list of possible manifestations by Levaditi and his associates[33] about the same time.

The appended case report is the first instance, recognized in this clinic, in which there was marked involvement of the lymph nodes outside of the regional inguinal group, and is, I believe, the first reported in detail in this country, though there is mention of two such cases in the reports of Kornblith,[31] and Martin and Bacon.[35]

Case Report.—Unit History No. 455929: An unmarried white male, age 45, presented himself at the Vanderbilt Clinic June 19, 1935, complaining of "swollen glands" of three weeks duration in the right groin. The family and past histories were irrelevant. He freely admitted frequent exposures with prostitutes but at first denied any form of sexual perversion. Twenty months later he admitted that his last sexual relation before his illness had been coitus buccalis with a prostitute about one month before the onset of symptoms. There had been no urethral discharge, and his first symptoms had been malaise, loss of appetite, pain in the right groin and in the lower back, and swelling of the right inguinal lymph nodes. After two weeks the pain decreased but the mass in the right groin remained.

Physical Examination revealed a small, thin man who did not appear acutely ill. His abdomen was generally resistant but without spasm or palpable masses. In the right inguinal region there was a 6 by 3 cm. movable, nontender swelling with its long axis in the direction of the spermatic cord which could be felt above it. It did not move with coughing or straining, and the skin over it was neither red nor hot. No scars or ulcers were obvious on the penis. Rectal examination was negative. Temperature 100.4° F. by mouth. Opinion was divided as to whether the mass was a hernia or an inflammatory mass, and, as the patient did not appear to be in a serious condition, operation was deferred.

Eleven days later he stated that on the previous evening he had been seized with violent generalized abdominal cramps. These had continued throughout the night, becoming milder in the morning. He had vomited once just before coming to the Clinic. His abdomen was slightly distended, tympanitic, and generally tender to palpation. The mass in the groin was possibly larger than when first seen but was not tender. Roentgenologic examination of the abdomen failed to show any evidence of intestinal obstruction. Temperature 102.2° F. by mouth. White blood cells, 12,200; differential P.M.N. 72 per cent; S.L. 20 per cent; L.L. 8 per cent. A preoperative diagnosis of inguinal adenitis was made, but with the possibility present of an incarcerated femoral hernia, the patient was operated upon at once. An abscess was encountered containing thick yellow pus and necrotic tissue. It was drained and packed with iodoform gauze and a silk tampon. Smears

and cultures were negative. Histologically, the tissue showed fat and fragments of lymph node in which there were foci of granular necrosis containing pyknotic nuclear remains and surrounded by zones of endothelioid cells generally arranged in radial fashion. Many mononuclear macrophages and a few multinucleated giant cells resembling Langhans' cells were present. No definite diagnosis was made.

Following the operation, his symptoms remained practically unchanged but his temperature undulated in a three day cycle from a peak of 104.0° F. per rectum to a low of 98.4° F. On the days when his temperature was elevated he complained of some abdominal pain and slight dizziness. The leukocytosis fell to 8,000 and the differential count remained the same. Widal, *B. melitensis, B. abortus,* and Felix-Weil agglutinations, chest films for tuberculosis, blood Wassermann, and abdominal films for calcified nodes were all negative. Finally two Frei tests were made and both were positive. His condition gradually became worse. He lost weight, had more severe abdominal pain, and retained the curious doughy feeling of his abdomen. On July 18 an exploratory celiotomy was performed but nothing was found other than some light brown peritoneal fluid, slight injection of the serosal vessels of the ileum, and many enlarged nodes along the aorta and right iliac vessels. The largest node measured 2 by 3 by 2 cm. and was soft but not fluctuant. It was removed and part of it used for guinea-pig inoculation and the rest kept for histologic study. This tissue showed partial obliteration of the node architecture by hyperplasia of the lymphoid and endothelioid cells, but no definite foci of necrosis. Postoperatively, his abdominal symptoms cleared up, the wound healed promptly, his temperature curve flattened out, and he was discharged six weeks after admission with his inguinal wound still draining. The guinea-pig was negative for tuberculosis. A small swelling appeared distal to the inguinal sinus early in September and this was incised and drained. Sections of the tissue removed at the time showed small foci of necrosis which were not caseous but had blood vessels and collagen fibers preserved in them. Rare multinucleated giant cells were seen and around some of the necrotic foci there were endothelioid cells. Both sinuses finally closed early in October.

On September 23, a small tender node appeared in the left anterior cervical region. It was watched carefully, and suddenly, on October 2 and 3, it more than doubled its size, still without signs of acute inflammation. The patient was admitted to the hospital and observed until October 16, during which time the mass slowly increased in size and became fluctuant at one focus. Clinically it presented the characteristics of tuberculosis of the cervical lymph nodes. An upper left neck dissection revealed several enlarged lymph nodes embedded in a mass of dense adherent fibrous tissue which extended much further forward than is usual in tuberculosis. In the center of the main mass a small amount of thick, gelatinous, pale-yellow pus, quite unlike tuberculous pus, was found. The wound was drained for 24 hours with a small Penrose tube and healed promptly. Pathologic examination again showed the same histologic picture as the tissue from the inguinal nodes, and guinea-pig inoculations from the pus and tissue were negative for tuberculosis. The patient was discharged on the seventh postoperative day, and after two weeks in a convalescent home returned to work. During a 17 month follow-up he has remained in fairly good health, having only occasional periods of malaise. In August 1936, he had an acute anterior cervical cellulitis which was treated privately. It subsided quickly and was probably related to very bad teeth rather than to his original infection.

Summary.—A man with known exposure to possible venereal disease and with a positive Frei test developed, successively, inguinal lymph node enlargement, constitutional symptoms of muscle pains, abdominal pain, fever, malaise, dizziness, and loss of weight and appetite, abdominal and iliac lymph node enlargement, and finally cervical adenopathy.

Etiology and Transmissability.—Hellerström and Wassén[24, 25] reported the passage of the infectious agent of lymphogranuloma inguinale through a

Berkefeld filter, in 1931, and thus established its identity as one of the filtrable viruses. Their work was confirmed a few months later by Levaditi and his co-workers.[32] The virus is apparently harbored both in the mouth and in the genitalia. Coutts[9, 10, 12, 13] has even maintained, for several years, that it is primarily a mouth inhabitant and attributes the apparent increase in the incidence of the disease to a growth of sexual perversion resulting from economic stringencies and misguided efforts at birth control. Once in the body, the virus is taken up by the cells of the reticulo-endothelial and lymphatic systems and distributed by them.[12] The incubation period is unknown, having been reported as being as short as three days and as long as four months (Coutts,[9, 12] Sézary,[46] Hellerström,[22] Nicolas and Favre,[17, 38] and others). One infection appears to confer immunity for life, but no data have been presented yet, regarding how long or under what circumstances a patient remains infectious.

*Symptomatology.**—The primary lesion is usually a tiny, soft, painless ulcer, though multiple ulcers and herpetiform eruptions have also been described. It occurs most frequently on the glans or shaft of the penis, on the vulva, in the vagina, and in the mouth; less frequently the rectum, the peri-anal nodes, and the extremities may be directly infected. There may or may not be a watery urethral or vaginal discharge for a few days. The genital lesions heal spontaneously in a few days or weeks, while those in the mouth may become quite large and persist for months, probably because of infection of the lingual and pharyngeal lymphoid tissue. In these cases there is what amounts to a coalescence of the primary lesion with secondary lymphoid tissue lesions. In primary infection of the rectum also, the initial ulcers merge gradually with the permanent lesions. With the ordinary genital infection, the patient is often quite unaware of the existence of any primary lesion, and, in over one-half of the cases, not even the tiny, white, stellate scar can be found on examination.

More or less painful swelling of the regional lymph nodes and constitutional symptoms of malaise, anorexia, weight loss, joint and muscle pains, fever, headaches, and dizziness appear along with, or one to two weeks after, the primary lesion. Nodular erythemata, peripapilledema, and tortuosity and congestion of the retinal vessels have also been described, but visual disturbances have never been noted. These symptoms persist for two to four weeks and then subside, leaving the nodes swollen but painless. In another two to four weeks, there is an exacerbation of symptoms. The nodes enlarge still further, and the overlying skin becomes rugose, indurated, and congested, finally breaking down to form multiple sinuses which drain thick, stringy, yellow pus for months. At the beginning of this phase there are often symptoms of involvement of the group of nodes next central to those originally

* The following section is a mélange of observations in this clinic and data from the writings of Nicolas and Favre,[17, 38] Coutts, *et al.*,[9, 10, 11, 12, 13] DeWolf and Van Cleve,[16] Ravault, *et al.*,[43, 44] Canizares,[5] Curth,[14] David and Loring,[15] Kornblith,[30, 31] Homma and Chaglassian,[26] and others.

attacked, and the French writers consider symptoms of iliac node disease following inguinal adenopathy almost pathognomonic of lymphogranuloma inguinale. After drainage has been established, the symptoms usually subside and the patient feels fairly well. However, it is important to note that these symptoms may be extremely mild and may even escape the patient's attention entirely. This is particularly true in those women whose primary lesion lies high in the vagina or in the cervix. The inguinal nodes do not enlarge in these cases, for the lymphatic drainage from this region is to the deep pelvic nodes and perirectal tissues. Hence, the first visit to the doctor may occur years after the original infection when symptoms of proctitis, elephantiasis of the vulva, condylomata, multiple fistulae in ano, or most commonly, rectal stenosis, have appeared. Occasionally a male is seen for the first time with these symptoms, and examination of the groins will reveal the scars of an inguinal infection contracted as along as 20 years before. However, rectal infection in men is usually direct and the result of pederasty, and the patients come in the early stages complaining of constitutional symptoms and proctitis. Examination reveals granular inflammation and multiple ulcerations of the rectal mucosa, without the fibrosis and stricture of the late cases.

Any general discussion of the manifestations of the disease outside of the lymph drainage areas regional to the portal of entry is unsatisfactory because of our present lack of knowledge of these forms. They seem to occur principally as complications of the acute phase of the more severe initial infections, but it is known that the disease may remain latent in local areas for many years and there may be as yet unrecognized lesions which develop far from the original site of infection years after that episode. As has been stated, nearly every part of the body may be affected and the lesions may be fulminating, and even fatal, or they may be subacute or chronic. In the lymph nodes, the disease seems to be self-limited and subsides after some months of drainage, while the extra-lymph node lesions are practically permanent. The recognition of such manifestations will often be simply the result of exclusion of other diseases and a final recourse to the Frei test, although the presence of one of the more typical inguinal or oral infections will be a valuable clue in many cases. About all that can really be said at present about these generalized forms is that any symptom complex of a granulomatous nature which does not clearly meet the criteria of one of the usually considered diagnoses should be thoroughly investigated for evidence of lymphogranuloma inguinale. Only in this way will the full clinical picture of this disease be finally brought to light.

Laboratory Data.—The laboratory diagnosis of lymphogranuloma inguinale depends essentially upon the Frei test, an intracutaneous allergic response to an antigen prepared from the pus or diseased tissue of a patient with the disease. As ordinarily carried out, the test has had several shortcomings, the principal ones being lack of availability of the antigen for the general practitioner, variations in the potency of antigens, and an absence of foreign protein controls. Many modifications of the original technic have been introduced in answer to these needs, the most important of which, aside from improved

preservation methods, has been the use of antigens prepared from infected mouse brains and controlled by similar preparations of normal mouse brains. Grace and Suskind[21] have written extensively and enthusiastically of this method, while Strauss and Howard[49] have been equally vehement in condemning it. Both human and mouse brain antigens are available commercially, and apparently both can give satisfactory results under the proper circumstances. Hardly anyone, to-day, denies that the test is consistent and reliable, though there is still great disagreement over how much of a symptom complex can be safely attributed to lymphogranuloma inguinale on the basis of the Frei test. As with many other diagnostic tests, single positives or negatives mean little or nothing; repeated tests with different antigens should always be made. Furthermore, an unusual interrelationship with the Wassermann reaction must be guarded against in this disease.

It seems to be definitely established that in the more acute phase of the disease the Wassermann reaction may become positive for short and often repeated periods. To further complicate matters, many of these patients actually do have concomitant syphilis, as well as gonorrhea. For these reasons, both tests must be repeated until consistent results are obtained and an interpretation then made on the basis of these results.

Other technical adjuncts are of little value. There is usually a leukocytosis of about 12,000 white blood cells per cm. with the differential count remaining essentially unchanged. Sometimes there will be a moderate relative increase in polymorphonuclear neutrophiles or a slight eosinophilia. The spinal fluid may or may not show an increase in the pressure, cell count, protein, and globulin, and occasionally it will be possible to prepare a Frei antigen from it. Finally, the intracerebral inoculation of white mice is beginning to assume the importance of the guinea-pig inoculation in tuberculosis, though the technic and interpretation are more difficult owing to the nonspecific histologic nature of the lesions.

Microscopically, the typical lesions are small stellate foci of necrosis in which there are nuclear remains and fragments of connective tissue. Around these foci are collections of lymphocytes, plasma cells, palisaded mononuclear macrophages, and endothelioid cells lying in a greater or less amount of dense, vascular connective tissue. Giant cells are common and may resemble either Langhans' or Reed-Sternberg cells. Unfortunately, this picture does not possess any unique characteristics; within its range of variation it may simulate tuberculosis, Hodgkin's disease, syphilis, or subacute pyogenic inflammation. Most pathologists, to-day, feel that the greatest value of tissue examination in this disease lies in ruling out other diseases whose histology is specific.[47, 3, 1, 38]

Treatment.—To date, there has been no satisfactory treatment devised for any form of the disease other than the more or less localized lymphadenopathies. Radical surgical excision of the diseased nodes offers prompt and apparently permanent relief of the local symptoms, but care must be exercised not to remove too much of the lymphatic bed lest progressive occlusion of the lymphatics occur and lead to elephantiasis of the parts distal to the nodes.

Innumerable biologic and chemical agents have been tried and there are enthusiasts and severe critics for each. It must always be remembered that the disease undergoes spontaneous remissions and exacerbations and no favorable results can be attributed to any therapy unless they are obtained promptly and consistently and are lasting.[5]

Prognosis.—As long as the disease remains localized to the lymph nodes, the prognosis appears to be good. Once it has spread beyond these boundaries, the lesions last for life; the patient may not die *of* the disease but will certainly die *with* it.

SUMMARY

A case is presented of lymphogranuloma inguinale with severe constitutional symptoms and involvement of the inguinal, iliac, abdominal, and cervical lymph nodes.

A brief review of the literature shows the steady growth of the conception of the extent of the disease.

The symptomatology, laboratory findings, treatment, and prognosis of the disease are summarized. No treatment has been definitely shown to have any permanent effect.

An appeal is made for more general use of the Frei test in obscure disease syndromes.

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PILONIDAL SINUS

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THE clinical entity which has come to be known most widely by the term pilonidal sinus is regarded by many physicians and surgeons as a minor surgical condition: a furuncle of the sacrococcygeal region which is incised, drained, and all too frequently forgotten. It is described in the text-books of surgery as a minor surgical procedure and yet if we consider the percentage of recurrences as compared with many of the so-called major surgical operations and the results obtained from the present form of treatment, it would merit more consideration than has been accorded it in the past. The frequency of recurrence with its accompanying annoying symptoms of pain and discharge, together with the economic losses resulting from complete or semi-invalidism for variable periods up to two months or more, makes this condition a thorn in the flesh to doctor and patient alike.

The lesion has been described in the literature of the early nineteenth century, but it was not clearly differentiated until Warren,[1] in 1867, made the first thorough description, and Hodges,[2] in 1880, contributed the descriptive term "pilonidal," meaning nest of hair. We have subsequently noted that hair is found in less than half the cases, but this term, because of wide usage, has taken precedence over more anatomically descriptive terms such as sacrococcygeal sinus or coccygeal fistula. Much of the early investigative work has been done by Europeans (Tourneaux, Oehlecker, Lannelongue and others). Mallory,[4] in 1898, in this country, contributed a study of the caudal end of the embryo with this structure in mind. Later clinicians (Stone,[5] Rogers,[6] Hall,[7] Fox[8] and Gage[9]) have added to the accumulated knowledge of the past. In spite of this, the last word on the etiology and successful treatment has not been said. It remains for the future to decide the relative merits of newer methods of treatment as compared with those previously accepted, unsatisfactory though they may be.

Incidence.—The present communication is an attempt to present some of the essential and significant factors in the causation, pathology and treatment of pilonidal sinus as revealed in our material and in that presented in past and current literature. In a period of 11 years (1925–1936) we have seen 113 cases of pilonidal sinus in 96 patients. Of this number 84 patients were operated upon 101 times. Nine of the 84 were operated upon 25 times.

The frequency with which pilonidal sinus occurs can best be appreciated by briefly presenting its occurrence as revealed by statistical study in reports obtained from clinic and hospital groups. It should be made clear, however,

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that in the figures cited, the authors list as pilonidal sinus the so-called "sacral dimple" which is found in the new-born and which is considered a potential source of the pilonidal sinus as it is later recognized clinically, when it presents a well differentiated, infected tract. Table I(A) shows the fairly high incidence of defects in the sacrococcygeal region of children and adults. However, all of these individuals who show a defect in early and later years do not have a sinus tract, as illustrated in the occurrence of pilonidal sinus in only 3 to 4 per cent of the patients examined. The second part of Table I(B) shows the ratio of occurrence in general clinic and hospital admissions. Familial tendencies and the importance of trauma in the occurrence of pilonidal sinus will find as many adherents as opponents. Some series show a com-

TABLE I [6, 7, 12]

(A) OCCURRENCE OF SACROCOCCYGEAL DEFECTS

20–25 per cent of all children
33⅓ per cent of 300 consecutive births
39 per cent in 85 children under 4 years
23 per cent of all patients examined
20–30 per cent of all patients examined
3 per cent of adults examined have a true sinus

(B) INCIDENCE OF PILONIDAL SINUS

30 cases in 19,000 patients.....	1 : 600
30 cases in 19,000 patients.....	1 : 600
7 cases in 15,000 patients.....	1 : 2,143
288 cases in 481,384 patients.....	1 : 1,683
96 cases in 149,913 patients.....	1 : 1,561*

* Author's statistics.

paratively high incidence of familial occurrences, but in our series there were only five patients who reported a similar condition in an immediate member of their family. Two brothers are included in our series. Nineteen out of 96 patients list trauma as a factor and mention falls, kicks, riding on tractors, *etc.*, as the exciting agent. Sedentary occupations are reported as predisposing conditions, and students, office workers and others of sedentary habits have been cited as examples. Other authors disagree with this observation and state that the type of individual is more significant. This includes the heavy, obese, hypopituitary type where the skin glands are overactive and infection is frequent and persistent irrespective of the habits or work of the individual.

Age, Race and *Sex* reveal several noteworthy points and also leave unanswered several questions. Although the sacral dimple is very frequently seen in the new-born, the active, inflammatory, symptom-producing period of the pilonidal sinus is found in the third and fourth decades of life. That it persists into the later years of a man's life can be shown, but its first flare-up usually occurs during the latter part of the second to the early part of the fourth decade. The youngest patient operated upon was eight years old, the oldest 57, average 27.3 years. Other authors place the average age in the early twenties: Ferguson[10] (21–25), Rogers[6] (25), Smiley[11] (18–28). Of

the 96 patients observed, the peak appeared in the third decade when 55 patients, with a diagnosis of pilonidal sinus, in this age group were seen in the clinic. The first and last two ten year periods were about equal, although the duration of symptoms in those over 50 goes back 10 to 20 and even 35 years.

The occurrence, with regard to sex, is predominantly on the male side. Out of 96 patients 78 were males and 18 were females, a ratio of 4:1. The findings of other authors confirm this sex ratio.

All our patients were of the Caucasian race, and many reports state that this condition is confined solely to this race. The literature reveals, however, that the Negro is also afflicted, although to a much less degree. Breidenbach and Wilson[12] report three cases in their series, while Smiley,[11] and Fansler and Anderson[13] record one each. No reports have mentioned the occurrence of the lesion in the yellow, brown or red races. It opens an interesting field for speculation as to whether this is due to surveys being confined to the white race or to developmental or environmental factors being paramount in the production of pilonidal sinus.

Etiology.—The correct treatment of pilonidal sinus should, and must, rest on a clear conception of the factors responsible for its formation. On one point all authors are agreed: That it is of congenital origin. Beyond this, opinion is divided into two principal schools: Those who believe that it is due to an infolding of the epithelial layers of the skin; and those who assert that it is a remnant of the neurenteric canal. The proponents of these theories include men well known in both clinical and investigative fields (Lannelongue, Oehlecker, Bland-Sutton, Mallory, Ewing, Streeter, Stone and many others). Of the recent investigators, Fox,[8] and Gage,[9] each representing an opposing view and each having arrived at his conclusions by the study of serial sections of the human fetus, present the most recent available evidence.

Fox found ectodermal invaginations in the three and one-half month fetus lying just cephalad to the anus. These creases became pit-like in the five and one-half month fetus, and histologic sections showed that the epithelium was thicker in the areas of invagination. As older specimens were observed, the ectodermal invaginations became deeper until shortly after viability (27½ weeks) it lay directly over the coccyx. Regression began at 29½ weeks, and if these depressions persisted into postnatal life they formed the sacral dimple or sinus tract seen in the child or adult. Stone[3] has suggested that this process may be a vestigial remnant of the gland found in birds which is used in oiling the feathers. He has called it the "preen gland." It is located near the end of the spine, and is composed of several straight tubules which drain through a common duct lined with modified squamous epithelium. Analogous glands are found in reptiles and mammals and are called sex glands. They develop with maturity and may be regarded as secondary sex characteristics. If the parallel is carried further these remnants may develop in the human with the onset of puberty, thus accounting for the prevalence of activity of the pilonidal sinus in the latter part of the second and third decades.

Gage, as a result of his investigative work, believes that the true pilonidal

sinus is due to the persistence of the neurenteric or neural canal. Normally in the caudal end of the embryo that portion of the neural canal which is formed by the union of the neural folds and which lies between the skin and coccygeal vertebrae is obliterated by cohesion of its walls. Should this portion fail to become obliterated by cohesion, it will form a cavity which may be connected with the skin by one or more sinuses. It may be entirely isolated from the remainder of the neural canal or it may be connected with it by a patent filum terminal. The lining of the cystic cavity will retain the faculty of producing the primitive skin appendages—the hair follicle or shaft, and a rudimentary type of nervous tissue if a connection with the spinal canal remains. Gage has been able to demonstrate both types of tissue in histologic sections taken from excised pilonidal sinuses.

He believes that this is the origin of the true pilonidal sinus, and the sacral dimple is the result of an anterior pull on the overlying skin by the caudal ligament as the coccyx grows downward and curves anteriorly.

From the foregoing discussion it becomes apparent that it will make some difference in the treatment, depending upon which source of origin is recognized. According to Fox, a block dissection down to the sacrococcygeal fascia will remove all sinus elements, while if Gage's findings are accepted it may be necessary to strip the periosteum and even remove the coccyx.

Symptomatology and Pathology.—Grossly, the pilonidal sinus usually consists of a draining sinus which opens in the midline several centimeters cephalad to the anus. The surrounding tissue is frequently acutely inflamed or chronically indurated. A probe passed into the sinus tract leads cephalad and toward the sacrum. Often the number of sinuses may vary considerably with the amount of infection, and there may be openings for several centimeters to either side of the midline. The usual, uncomplicated pilonidal sinus consists of a single, midline sinus leading down to a cystic cavity lying in the subcutaneous tissue above the periosteum of the sacrococcygeal vertebrae. Hair may be seen protruding from the sinus opening, but this is not a frequent finding when infection has been present any length of time. In this case the squamous epithelium has been replaced by a chronic granulation tissue and the deeper tissues contain collections of lymphocytes, plasma cells, foreign body giant cells or groups or polymorphonuclear leukocytes. Only 56 per cent of our cases showed hair follicles or shafts on gross or microscopic examination. Masson[14] reported 35 per cent in 86 cases, and Owen[15] lists ten out of 40 cases.

Bacteriologic studies were carried out on only eight cases, chiefly with pus from acutely inflamed, unopened cysts. There was no growth on four cultures and the remaining four showed *Staphylococcus aureus* and *albus*, a gram-positive rod and diplococcus. The number of sinuses varied from none to ten, the average being two. Inflammatory reaction was present in well over 90 per cent of the cases.

In two patients an extension of the pilonidal sinus was noted. In one instance a dermoid cyst was found lying anterior to the coccyx and a second

tract extended up along the anterior surface of the sacrum to the promontory. The coccyx was removed. The secondary tract was diagnosed as a pilonidal sinus and histologic sections revealed rudimentary glial cells. In the second case there appeared to be a cord extending from the pilonidal sinus into the periosteum of the sacrum. It was decided that it was a vein and it was cut. Follow-up revealed that the patient subsequently developed a recurrence. Reports have been published in the literature of meningitis occurring in individuals with pilonidal sinuses.[22] Opponents of the neurenteric theory state that these individuals have a spina bifida occulta and the lesion is located higher than the usual site of occurrence.

The symptoms of pilonidal sinus are quite characteristic of inflammations elsewhere—pain, redness, heat, and swelling with an irritating, seropurulent discharge. Drainage of the acutely inflamed cyst, whether by incision, trauma, or spontaneous rupture, gives relief from the pressure symptoms, but this is often followed by the aggravating, continuous or intermittent drainage which causes excoriation of the surrounding skin. Subsidence may, and frequently does, occur with a remission of symptoms for variable periods of time. In our series the symptoms ranged from one week to 35 years, with an average of six years. Of 96 patients, 57 had received no previous treatment, although in many the cyst had ruptured spontaneously. Of the remaining 38 who had received treatment, 34 had been incised, ten had had excision operations, in two the tract had been injected with a sclerosing solution and in one patient it had been curetted.

Treatment.—The incisional method is only palliative; useful merely in promoting drainage and subsidence of the acute inflammatory phase. As a method of permanent relief, it has been discarded and the excision of the sinus tract and cyst is the generally accepted procedure at the present time. The controversial points are chiefly around the amount of tissue or bone removed, the methods of closure and postoperative care. Of the 101 cases operated upon, 87 were treated by excision, and it is this group that is particularly considered. Before describing the methods of operating, three preoperative factors will be discussed briefly: Skin preparation; anesthesia; and the use of dyes to visualize the sinus tract.

The area in which the lesion is located is a fertile field for contamination from the gastro-intestinal tract, and perspiration and body heat increase the conditions favorable for bacterial growth. Preparation of the skin should receive meticulous care prior to operation and during the period of wound healing. Frequently the skin is excoriated for some distance around the sinus openings and operation is best delayed until this has been cleared up. In our series the standard antiseptics such as iodine, picric acid and merthiolate have been used, the tendency being to employ the latter two in recent years. Since gentian violet has been used successfully in cutaneous infections and in preparing the body surface for wide operative fields as in thoracic surgery, better preparation might be obtained by the use of this agent.

The inhalation anesthetics have been used predominantly in this type of

operation, but during recent years there has been a growing tendency to use spinal[15] or local[7] infiltration, notwithstanding the fact that an infected field is being invaded. Forty-three of our cases were anesthetized with ethylene, four with ether, eight with nitrous oxide, one with cyclopropane, two with spinal, and four with local novocain infiltration.

Dyes and colored solutions have been used for years in the treatment of pilonidal sinus. Most surgeons feel that injecting the tract with methylene blue facilitates removal and gives an added sense of assurance that the tract has been entirely eradicated. Opposed to this view is a smaller group represented chiefly by Rogers and Hall,[7] who believe that the use of dye is dangerous and detrimental to complete excision because of the false sense of security which it gives. Weeder,[16] believing that many recurrences are due to failure to follow the tract down to the periosteum and coccyx, advocates the use of a radiopaque solution, such as lipiodol, for visualization of the sinuses and possible connections with the sacrum or spinal canal. Seventy-five out of 87 cases operated upon by the excision method in our series had the sinus tract injected with methylene blue, and it was recorded on the operative report whether or not dye-stained tissue was cut across. Breidenbach and Wilson,[12] in their large series of cases (287), reported little difference in the cured cases, irrespective of the use of methylene blue.

The excision method, as carried out in most clinics, utilizes a wide, elliptical incision which includes the tract, and a block dissection of the diseased tissue down to the sacrococcygeal fascia. The defect may then be left open and packed, allowing the defect to granulate in; or the edges may be approximated with deep retention sutures and iodoform gauze packed between the wound edges. The third method is primary closure with or without drainage. Plastic procedures may be used, such as that described by Lahey.[17] Methods other than excision have been advocated but have not been generally adopted. Cutler and Zollinger[18] used a modified Carnoy's solution (chloroform, absolute alcohol, glacial acetic acid, ferric chloride) in various fistulae, sinuses and cerebellar cysts. They laid open several pilonidal sinuses and cauterized the tract with the sclerosing solution. Smiley[11] reported three cases, two of which were well at the end of six months. Rogers and Hall[7] reported only one case treated in this manner and abandoned the method because of repeated sloughs and residual sinuses.

Probably the most recent method is that described by Stanton,[19] in 1932, and applied by Rogers and Hall[7] to a series of 50 patients over an eight months' period. The treatment is ambulatory, requires a very brief period of hospitalization, and the patient is followed in the clinic. It consists of removing the sinus tract and a minimum of surrounding tissue, under local anesthesia, without sacrificing the overlying skin. The electrocautery is used, the defect packed, and the diseased tissue which remains is removed on subsequent visits in the same manner. No dyes are employed and hemostasis is controlled by packing and thromboplastin.

Results.—Tables II and III show the results obtained in a series of 87

cases treated by the excision method. These operations were performed by 24 different surgeons. Of the 87 cases operated upon, 61 were traced by letter or made a return visit to the clinic. Of the number traced, 22 recurrences were noted, a recurrence percentage of 36. The average postoperative

TABLE II

METHOD OF TREATMENT

Total number of cases	101*
Excision method	87
Primary closure with no drainage	2
Primary closure with drainage	68
Partial closure over gauze pack	6
Wide open packing	11
Incision method	14
Total cases drained—excision and incision	99
Methylene blue injected into sinuses	74
Average postoperative days in hospital	14.2

* Although 113 cases were listed, 12 of these were in new-borns in which only a sacral dimple was present. These cases were not considered as true pilonidal sinuses and have therefore not been included in the above tables.

stay in the hospital was 14.2 days. Of the 87 cases, 35 healed by primary and 52 by secondary intention. A second operation was required for hemorrhage, excision of a secondary tract, skin grafting and the drainage of an abscess. An appendicectomy, a herniotomy, and the drainage of a subdiaphragmatic abscess occurred in three patients while they were being hospitalized. Hemorrhoids and fistulae were operated upon in several instances while the pilonidal sinus was being treated.

TABLE III

RESULTS OF EXCISION METHOD AS REVEALED BY FOLLOW-UP

	Excision. Primary Suture. No Drainage	Excision. Primary Suture. With Drainage	Excision. Partial Closure. Gauze Pack	Excision. Wide Open. Packing	Total Cases
Number of operations	2	68	6	11	87
Number of follow-ups	0	45	5	11	61
Number of recurrences	0	14	1	7	22
Percentage of recurrences	0%	31%	20%	63%	36%

Table III shows the methods employed, with the number of operations performed by each method, and the number of cases with recurrence following the various procedures. The majority of our cases were treated by excision, primary suture and drainage; but the highest percentage of recurrences occurred in those cases which were excised and packed wide open. This method was used chiefly in cases which had been unsuccessfully operated upon.

The percentage of recurrence in our series is higher than those reported by other authors who have written upon the results of the excision method

(Rogers and Hall—30 per cent (first series); Rogers and Hall—34 per cent (second series); Rogers and Hall[7]—0 per cent (third series); Breidenbach and Wilson[12]—23 per cent; Owen[15]—8 per cent; Cattell and Stoller[20]—23 per cent; Ferguson[10]—17 per cent; and Glenn[21]—28 percent).

Discussion.—It is interesting to note that our figures compare more favorably with those reported in the first and second series of Rogers and Hall.[7] These authors, at the completion of their first series, drew up a strict regimen by which all the patients in their second series were to be governed. In spite of this, the percentage of recurrence was higher, and they sought another method by which the end-results would be improved and the period of hospitalization and loss of time from work would be reduced. In the application of the method of Stanton,[19] previously described, the patients were not selected according to the severity of the condition, but were taken as they presented themselves: namely, recurrences as well as new cases. Of the 50 patients operated upon during an eight months' period, 29 were far enough along to judge the results. In this group there were no recurrences and the average stay in the hospital was one day. All the cases were operated upon under local anesthesia.

Ferguson[10] has reported a series of pilonidal sinus cases in which he used local anesthesia, injected the sinus with methylene blue, and preserved as much of the skin and normal tissue as possible. Closure was complete and without drainage. The patient was placed on his back for an hour, in order to effect hemostasis, and was then able to continue his work without further loss of time. The skin sutures were removed on the third day. Twenty-four cases, thus treated, were followed for at least one year, with recurrence in but one instance. In a previous series in which incision or excision and packing had been employed, Ferguson's recurrences varied from 50 to 90 per cent.

Owen, in a series of 40 cases, had two recurrences. Twenty-three, or 57.5 per cent, were treated by the excision and wide open packing method, the remaining 17 by excision and primary closure with drainage or partial packing.

In reviewing the 22 cases in our series in which recurrence was noted, 14 had received previous treatment, approximately 65 per cent. The highest percentage of recurrences occurred in those cases in which the defect was allowed to heal by wide open packing and granulation. In many of these patients, in whom this method was used, there had been marked infection, and primary closure was thought to be inadvisable. These wounds required frequent attention and the healing was tedious.

Summary.—(1) During a period of 11 years, 96 patients have been observed, with a condition which has been diagnosed as pilonidal sinus. Several of the patients have been treated twice or oftener for a recurrence of the original condition, making a total of 113 instances in all.

(2) Although many new-born children and infants show a dimpling or depression over the sacrococcygeal region, nevertheless, the presence of a

true sinus tract in adults is limited to about 3 or 4 per cent of the population. Males show a considerable predominance in the incidence of this condition, and the age average falls chiefly in the third decade.

(3) The theories of origin are confined chiefly to whether the cyst or sinus arises from the neurenteric canal or is the result of ectodermal invagination. We believe that clinical and experimental evidence lends more support to the former theory.

(4) The pathology and symptomatology in the average case are similar to those of a chronic inflammatory process which has undergone an acute phase with periods of chronicity and subsequent exacerbations.

(5) Treatment consists essentially in the complete eradication of the sinus tract without the sacrifice of any more normal tissue than is necessary. We believe that the injection of the sinus with methylene blue is conducive to a false sense of security and the needless sacrifice of normal tissue.

(6) Recurrences are frequent and are due to several factors: Failure to remove the sinus tract; operation for excision in the face of acute infection; poor hemostasis; and the presence of dead space with subsequent hemorrhage and infection.

CONCLUSIONS

Our conclusions, in general, agree with those noted in the body of the paper. However, there are several facts which are worthy of reiteration for the sake of emphasizing their importance in the treatment of pilonidal sinus.

We believe that Gage presents the more embryologically correct explanation of the occurrence of the true pilonidal sinus and that his evidence fits in with the clinical findings. The occurrence of cysts or sinuses which are not true pilonidal sinuses may be explained by the data presented by Oehlecker and Fox.[8] A connection with the sacral and spinal canal may not always be demonstrable due to the obliteration of the connection which takes place. In this instance the injection of lipiodol should be attempted in an effort to visualize the extent of the sinus tract and its relationship to the bony structures or the spinal canal. The relationship of the sinus tract to the sacrococcygeal vertebrae will probably be found to be higher than it is at present thought, although it is doubtful whether it will require the removal of the coccyx in 30 per cent of the cases, as has been suggested.[16]

The use of a dye is of questionable value and depends upon several factors. If given with sufficient force to penetrate the entire tract, it may burst through and stain the tissues, as has been demonstrated on histologic sections.[7] On the other hand, if insufficient pressure is used, the dye will not penetrate the smaller radicles and a false sense of security will be given. The value of the dye has been overestimated and its disadvantages minimized.

The essential factor in recurrence, so far as we have observed it, is the presence of infection in the tissue and the formation of sinuses arising out of this infected tissue. We are not discounting the remnants of the sinus tract which may remain, but feel that this fact has been emphasized to the

point where too large amounts of normal tissue have been removed, with the opportunity for bleeding, infection and the development of dead space. The first step in the reduction of this factor is the recognition of the need to postpone operation where acute infection is present. The principle is observed elsewhere in the body and should be particularly true in this region. Incision is the method of choice under these circumstances. Skin preparations should be rigidly observed and the use of gentian violet has been suggested as a more satisfactory means of securing this result as evidenced by its success in cutaneous infections elsewhere.

In their desire to completely excise the tract, many operators remove too large a block of tissue. It has been shown embryologically and by pathologic examination that the majority of sinuses are in the midline. A small block of tissue will remove the pathologic tissue without exposing the surrounding areas to contamination, and the lateral tracts which are found are very frequently the result of infection burrowing to the surface.

We believe that the wound should be closed if the surgeon feels reasonably sure that it is not grossly infected. Hemostasis and obliteration of dead space should be effected. If drains are used they should be inserted at the upper angle or between the stitches, not from the lower angle. If the wound is grossly infected then wide open packing is the method of choice, providing the postoperative care can be carefully given and closely followed. The repeated cautery and packing method advocated by Rogers and Hall[7] cannot be used in this clinic because of our inability to follow the patient as closely as the method requires.

The use of pressure over the sacrococcygeal region for one hour postoperative, as suggested and practiced by Ferguson,[10] is beneficial in securing hemostasis and obliterating dead space. We believe that in many of the simpler types of pilonidal sinus it would promote faster healing, lessen the danger of infection, and materially reduce the period of hospitalization, by having the patient up on his feet sooner. Strapping the buttocks together will take some of the tension off the sutures and will also prevent too free motion when walking.

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PILONIDAL SINUS

OBSERVATIONS ON ONE HUNDRED FORTY CASES TREATED BY CAUTERY EXCISION

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THE purpose of this paper is to present the experience we have gained through treating a series of cases of pilonidal sinus by the method described by one of us,[1] in 1935, as "Cautery Excision." The present report comprises 140 unselected hospital cases, both new and "recurrent." In it no distinction between previously treated and untreated cases will be made, as no significant difference in their behavior under the present method of treatment has been observed. The earliest excision was in July, 1934, and the latest in April, 1937. During this period, while the practice of hospitalizing patients with pilonidal sinus has been giving way to out-patient management, the ratio of male to female patients has increased from 2:1 in 1934 to 14:1 in 1937. Of the 140 patients, 102 were male and 38 female, or a ratio of practically 3:1.

Two patients were Negroes, the rest white—a high Negro incidence in a clinic almost exclusively white.

Ages at the time of treatment were chiefly in the latter part of the second decade and in the early part of the third, the youngest being age 15 and the oldest 42.

Although the disease varied greatly in extent and severity, there was no case of extension into the neural canal, sacral hiatus, sacrococcygeal joint, or beneath the sacral aponeurosis. No case of cancer associated with pilonidal sinus was encountered.

Excision was not undertaken in the presence of acute abscesses. These were drained by incision in the midline or by dilatation of a midline sinus-opening at least three weeks before excision was performed.

As far as possible the same surgeon who performed the excision also took care of the dressings. When this rule was disregarded the patient was likely to suffer. Ninety-seven patients were treated entirely by the authors, 30 by members of the resident staff under our supervision, and 13 by other members of the visiting staff.

All of the patients were ambulatory throughout their treatment except 14 whose excisions were accomplished under a general anesthetic, necessitating a brief period of hospitalization, usually 24 hours, and four who returned to the hospital for various complications.

RATIONALE OF CAUTERY EXCISION.—In attempting to evaluate the various

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methods of treating pilonidal sinus it should be clearly recognized that whether the condition be simple or complex, permanent cure depends on the fulfillment of only two major principles: (1) Its cause, *i.e.*, hair, ectopic epithelium and infected scar tissue, must be removed; and (2) the resultant wound must heal solidly, *i.e.*, without dead space or infected scar tissue.

The former of these two principles usually presents few difficulties and may be accomplished in a variety of ways. Very radical block excisions, however, have been abandoned, as a better understanding of the morphology has shown them unnecessary,[2] and more careful follow-up has shown them too prone to fail of cure. Various chemical and physical agents have been suggested to destroy the ectopic epithelial cells which constitute the congenital variation associated with the later development of pilonidal sinus. These suggestions fail to take into account the possibility that the infected scar tissue present in every pilonidal sinus as a result of trauma and infection, rather than the ectopic epithelial cells, may be the actual cause of the sinus. As a matter of fact, what evidence there is tends to show that ectopic epithelial elements alone usually do not cause sinuses. If they did, we should expect that: (a) As many females as males would have sinuses, since the congenital defect is not known to be dependent upon sex; (b) sinuses would uniformly appear in early life, having only a congenital cause; (c) complete excision of ectopic epithelial elements would always result in cure; and (d) ectopic epithelial cells would always be demonstrable in tissue excised for "recurrent pilonidal sinus." The facts are that: (a) Males outnumber females at least three to one; (b) sinuses sometimes do not appear until age 50; (c) of 140 radical block excisions reported by one of us,[2] 30 per cent failed to cure the sinus; and (d) serial microscopic sections have shown ectopic epithelial elements in less than 10 per cent of "recurrent" sinuses[2]—the true figure being nearer 1 per cent, in our opinion. On the other hand, the last two items of evidence are enough to prove that chronically infected scar tissue alone is quite capable of keeping a sinus draining. Thus we believe that though cure cannot be expected unless all of the diseased tissue be excised, excision satisfies only one of the two essential principles.

Granted that some sort of excision be necessary, the method of its accomplishment should be predicated upon the factors governing the healing of the resultant wound, that is, if these wounds will heal more safely by second intention than by first, there can be no point in using a technic the only recommendation of which is that it permits healing by first intention. Many surgeons who would not think of courting failure by a primary closure of the wound still handicap themselves by performing a comparatively blind, bloody excision, when by the use of the proper cautery technic they could accomplish the same end more quickly, more accurately, more conservatively, and more easily in a bloodless field. What, then, is the case for primary closure?

In the first place, primary closure often succeeds; in our experience about 50 per cent of such attempts (in selected cases) have resulted satisfactorily. Secondly, a *successful* primary closure does away with the more or less pro-

longed course of dressings which are essential to the success of second intention healing—in other words, it is less irksome to the surgeon as well as the patient. Granting, however, that first intention healing is desirable, it still does not follow that the best way for all pilonidal wounds to heal is by first intention.

It has been our experience that failures due to defective wound healing outnumber those due to incomplete excision at least ten to one. Few parts of the body are so exposed to bacterial contamination, minor trauma, and unavoidable muscular motion. In common with other median raphes, the tissues in this region have a natural tendency to draw apart when longitudinally incised, leaving a good-sized cavity even though no tissue has been removed. When one considers the fact that sepsis—chronic, acute, or both—is the factor which actually brings the pilonidal cyst to the attention of the patient as well as the surgeon, it becomes apparent that ordinary measures, which would succeed in other parts of the body, must frequently result in failure when applied to this type of lesion.

Of course these unfavorable factors are present in varying combinations and degrees depending on the exact location and extent of the wound. A wound made by the aseptic excision of a small, uninfected cyst overlying the upper coccyx, for example, might be carefully sutured with a fair expectation of primary union and solid healing. But even here, hidden, dead space and postoperative contamination would cause an occasional failure. We have twice seen persistent sinuses result from clean coccygectomies for painful coccyx, and Ferguson,[3] who has brought the technic of primary closure very near perfection, has had failures from these causes. On the other hand, failure is to be expected from suturing a wound left by the excision of an extensive, infected sinus tract with openings near the anus. In fact, we have seen many cases in which complete, conservative excision by sharp dissection would have been almost impossible because of lack of visibility, and suture without dead space resulting entirely impossible.

In view, therefore, of the many obstacles to the healing of wounds in the coccygeal region, we are now satisfied that most pilonidal sinuses can best be excised by the cautery technic, the wounds kept packed and allowed to heal by second intention, and that regardless of the technic of excision, if constant observation of the healing wounds be omitted, either through neglect or as a consequence of primary suturing, the solid healing of even a simple wound must be largely a matter of chance.

Technic of Cautery Excision.—*Materials:* "Post" cautery equipped with point No. A 4.

Four double hooks (or a small self-retaining rake retractor if no assistant is available).

Toothed tissue forceps.

Tuberculin syringe equipped with a hypodermic needle.

Five cubic centimeter syringe equipped with a hypodermic needle.

Sixty cubic centimeters of a 1 per cent novocain solution containing 0.2 cc. of 1–1000 adrenalin solution.

Sterile gauze sponges.

Sterile mastoid sponges ("Hibb's").

"Thrombol" (thromboplastin), or other local coagulant.

Butesin picrate ointment.

1 per cent aqueous mercurochrome (or hexylresorcinol).

Preparation of Patient.—The sacrococcygeal region is dry shaved and the skin cleaned with alcohol and ether. The patient lies on his right side with the knees drawn up and a comfortable pillow under his head.

Arrangement of Operating Room.—The surgeon sits with his head on

FIG. 1.—Midline incision with skin held tense to avoid charring. This incision may go entirely through the diseased area, exposing the pre-aponeurotic areolar layer and dividing the specimen longitudinally into halves, or simply into the hair-nest, as in Fig. 2.

a level with the operative field, with a strong light coming over his shoulder. The instrument table is within reach of his left hand. The assistant (if any) stands on a platform in front of the patient where he is in a position to retract the left buttock upward.

Anesthesia.—Using the tuberculin syringe, an intradermal wheal is made along the midline, going both sides of each sinus opening, and extending one inch above and below the limits of the proposed incision. Then, using the 5 cc. syringe, novocain is injected just beneath the skin to either side of the midline to a distance of about one inch.

Skin Incision.—With the cautery at maximum heat, and with the skin held on the stretch, an incision is made along the midline (Fig. 1), laying open all midline sinus tracts, and extending into or through the hair-nest.

Retraction.—The skin edge is grasped with tissue forceps and undercut with the cautery for 1 cm. along both sides of the incision (Fig. 2). The

upper (left) skin edge is then seized with double hooks, which are held up by the assistant. If no assistant is available, the self-retaining rake retractor is used instead.

Excision.—The surgeon, holding the cautery and a sterile sponge in the right hand, and tissue forceps in the left, now grasps the fat to the left of the midline, draws it strongly down and towards him, and with light touches of the cautery divides the tense tissue between the under surface of the skin and the outer surface of the hair-nest (Fig. 3). As the division goes deeper,

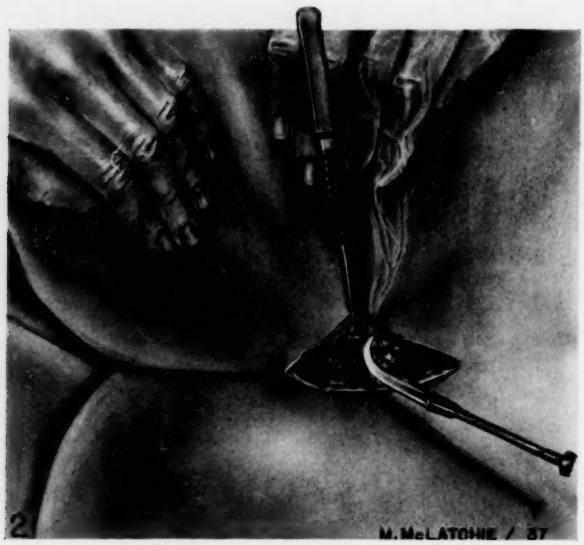


FIG. 2.—Hair-nest laid open through midline incision. Skin edges being undercut just enough to allow application of double hooks.

the forceps grasp the specimen farther in under the skin edge and roll it forward and downward, always keeping the tissue under the cautery-blade tense. As soon as the outermost limit of the diseased tissue has been passed (usually within 2 cm. of the midline) the cautery is directed anteriorly until the areolar tissue which covers the sacral aponeurosis is reached. This tissue constitutes a definite plane of cleavage, particularly at the cephalad end of the dissection, and it is left intact. The left half of the specimen is now peeled away from the areolar-tissue floor of the wound and removed (Fig. 4).

The right half of the specimen is excised in the same way, after applying double hooks along the right skin edge (Figs. 5 and 6). These double hooks may be held by the assistant, but usually their weight alone furnishes sufficient countertraction and exposure.

Hemostasis.—The heat of the cautery blade automatically prevents all venous oozing, but does not seal the small arteries, three or four of which are always encountered. As soon as a bleeding vessel is reached, all progress should be stopped, the wound sponged, and the bleeding point accurately touched with the flat of the cautery tip. If the cautery simply burns the

vessel back farther without sealing it, the heat must be reduced and the maneuver repeated. Progress must not be resumed until the bleeding has been stopped.

COMMENT ON TECHNIC.—The procedure described above is in no sense a destruction of diseased tissue by cauterization but a careful excision, with the cautery used as a knife, carried out under complete visual control, in a com-

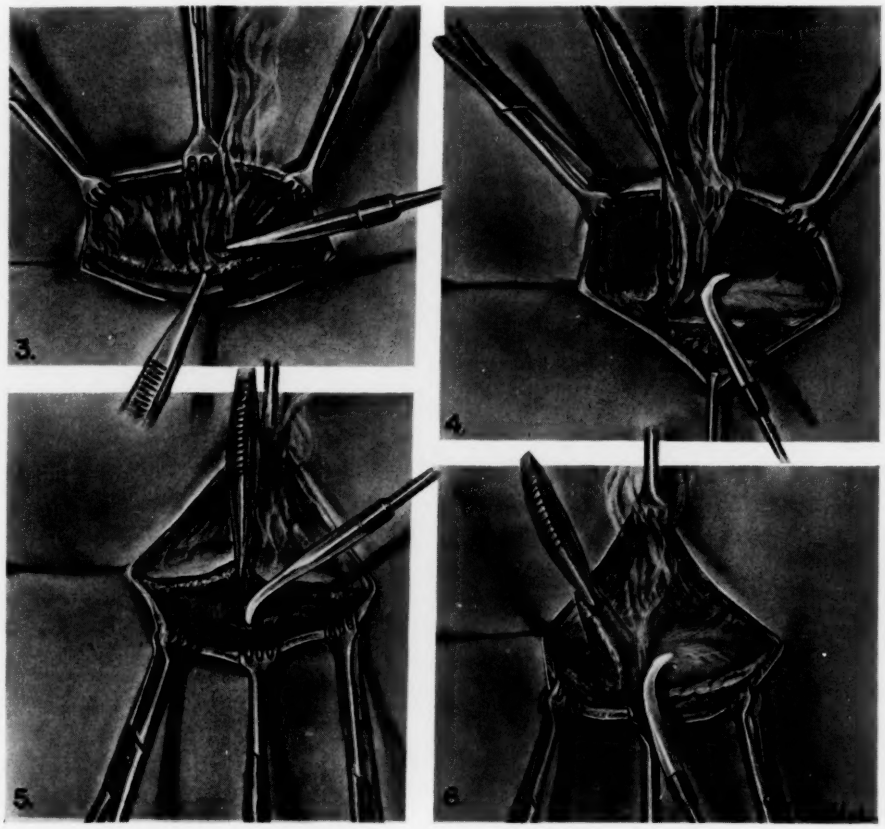


FIG. 3.—Skin edge retracted, left half of specimen drawn downward as it is dissected away from normal fat by light strokes of the cautery blade.

FIG. 4.—Left half of specimen (in thumb forceps) dissected free from lateral fat, and now being lifted clear of its fascial bed. The surface of the sensitive sacral fascia is seen shining through a protecting layer of insensitive areolar tissue.

FIG. 5.—Left half of specimen removed, and right half being dissected away from lateral fat in the same manner.

FIG. 6.—Right half of specimen almost removed. Sketches fail to indicate forward curve of coccyx which, if followed too far, will make distal end of wound unnecessarily deep. Note absence of charred tissue. The wound will be packed open.

paratively bloodless field. Three points should be particularly emphasized: (1) The cautery must be of the proper type and in good working order; (2) the surgeon must avoid boiling and frying the tissues; and (3) the hemostasis must be complete and effected as one progresses.

We have carried out cautery excisions both with and without aseptic precautions, and we can see no difference in the behavior of the wounds. We

continue to do the excisions in the Out-Patient Department in every case in which local anesthesia will be effective. We have learned, however, that the usefulness of local anesthesia is limited by the presence of extensive scar tissue, and prefer to operate upon such cases under a general anesthetic. In 148 excisions the anesthesia was local in 134, ether in eight, nitrous oxide in two, spinal in two, and evipal in two. In the average case we plan to take about ten minutes injecting the novocain and ten minutes to accomplish the actual excision. We have not yet needed more than 60 cc. of 1 per cent novocain.

Unless guided by other obvious landmarks, such as induration, we begin our midline incision 1 cm. below the lowest opening and carry it 1 cm. above the highest one, except in cases having only one opening, when it is carried 4 cm. above it. The relative size of the wounds was noted as large (over 8 cm. long) in 33, small (under 5 cm.) in 35, and average in 80. Accessory openings as far to the side as 2 cm. can easily be reached from the midline by undercutting, which is carried 0.5 cm. beyond them. When the disease extends farther than this we do not hesitate to make a second excision at right-angles, and it has been our experience that this cross incision is usually healed before the longitudinal one. This was done in five of our present cases, though it should have been done in more, and in only one was it necessary later to excise a corner of skin which persisted in sticking up and becoming epithelialized on its under surface.

Though desirable, it is not always essential to remove every bit of scar tissue. As our experience has increased, we have become more conservative in the amount of tissue removed, having gained greater confidence in our ability to recognize and remove it later, in the course of a dressing. In four cases we have deliberately left scar tissue which would have been awkward, but not impossible, to excise. All four patients, however, were cured and are free of symptoms, one more than two years and the others about one year after operation. The healing times, in round numbers, were 8, 13, 7 and 7 weeks, respectively. One patient in whom the scar tissue extended 8 cm. from the midline showed redness and tenderness of the overlying skin for about ten days, which, however, gradually subsided. On examination two years later the induration had entirely disappeared. In each instance the scar tissue that was left in was a considerable distance from the infected focus of either the main pilonidal abscess cavity or of any of its accessory drainage tracts, and appeared to be homogeneous "healthy" scar tissue containing no chronic granulation tissue or débris. The decision to leave such scar tissue *in situ* commits one to nothing, since it can be removed at any time during the process of healing when its presence appears objectionable, and we often prefer to leave it, provisionally, rather than make an unnecessarily extensive initial wound.

We now believe that it is almost never necessary to expose the surface of the sacral fascia in order to excise all of the diseased tissue. The floor of the wound should be the layer of pinkish areolar tissue through which the

glistening white surface of the fascia can be seen and felt. Near the lower end of the coccyx this guiding layer merges with a ligamentous structure which must be cut across arbitrarily, and if diseased tissue has extended below this point[4] it will be missed. Whether its presence be detected or not, we prefer to leave such diseased tissue for removal at a second stage procedure rather than to approach so close to the anus at the first operation.

Such a condition was present in ten of the cases in this series, in eight of which the downward extension was recognized at the original examination. In the other two it was overlooked even during excision, and these two cases are accordingly considered as failures. In two cases all of the diseased tissue was excised at one sitting, in four a two stage excision was carried out, and in the other four the second stage remains to be undertaken. When a low opening is seen, it must of course be distinguished from fistula-in-ano.

Two Stage Excision.—When extensive lesions are characterized by having one or more openings near the anus we believe the excision of all diseased tissue in one step to be unwise, even though the end-result may be satisfactory, since it produces a cloaca-like cavity too long for its depth and very difficult to keep packed. The sides of such a wound are flaccid and redundant when the thighs are extended, so that they either gape apart or curl in, with much rubbing together of the wound edges. The gross fecal contamination of so large a wound results in unnecessary pain, bleeding, irritation, and delay in healing. Excerpts from the records of two patients, unfortunately treated in this way, may be instructive.

Case 1.—E. F. D., male, age 24. Operated upon March 12, 1935: Cautery excision. Spinal anesthesia. Midline incision through multiple sinus openings over coccyx, meeting scattered bunches of hair. Extensive scar tissue encountered for 2 cm. on either side of midline extending downward almost to anus. Sinus tracts and scar tissue excised down to sacral fascia, a few sphincter fibers being exposed at lower end of wound.

April 3, 1935: Lower half of wound foul, bleeding and painful.

April 7, 1935: Lower end bleeding and sensitive. Almost impossible to keep pack in.

May 4, 1935: Filled in and starting to epithelialize. Scar irregular with lipped edge on left and considerable deformity.

May 18, 1935: Scar macerated for two inches by stiff hairs growing from rolled-in skin edge opposite. Must be kept shaved.

July 6, 1935: New epithelium chafed off near lower end. May need plastic to smooth scar and prevent this.

July 27, 1935: All healed and solid, 19 weeks, four days.

Case 2.—G. N., male, age 17. Operated upon December 13, 1934: Cautery excision. Ether anesthesia. Midline incision from level of an accessory opening over the sacrum to just above anus where hairs were seen protruding from a small sinus opening.

December 22, 1934: Still foul. Bleeds from lower end when pack removed.

January 5, 1935: Almost impossible to keep lower end packed. Quite impossible to keep it clean.

January 7, 1935: Very bloody and sensitive.

January 26, 1935: Needs shave but skin too abraded.

February 2, 1935: Clean and comfortable for first time.

March 6, 1935: Small granulating area at lower end. Still irritated.

March 23, 1935: Dime-sized area at lower end still lacks epithelium.

April 6, 1935: Healed throughout, 14 weeks, one day.
April 12, 1936: Soft scar. No symptoms.

At the first stage the diseased tissue in the posterior perineum is deliberately ignored. The transverse fold of tissue dividing the plane of the back from the plane of the perineum is easily seen with the thighs extended, but is less marked when the thighs are flexed. This fold should not be divided. In borderline cases, however, we have sometimes saved the patient a second stage by tunneling under it for a short distance when this maneuver would suffice to remove all the diseased tissues. The cavity resulting from the first stage excision is kept packed and allowed to heal except for the sinus remaining at or below its lower end.

We believe it is important that the first wound should have been healed for several weeks or even months before the second stage is undertaken. The three appended case reports illustrate this point.

Case 1.—H. K. Seven weeks after first stage, when wound was filled by granulation tissue but not yet epithelialized, the second stage was carried out, creating a small cavity whose upper wall was formed by the granulation tissue of the healing wound. Within two weeks all the granulation tissue of the upper wound melted away, leaving a single long shallow cavity which took 14 weeks and two days to heal. The final result was good.

Case 2.—C. D. Wound of first stage was allowed to stay healed for 12 days. Within two weeks of the second stage most of the upper wound had broken down and melted away as if by a cytolytic ferment. Final healing took 12 weeks and two days. The final result was good.

Case 3.—A. R. Three months was allowed to elapse after the first wound had healed before undertaking the second stage. The first wound stayed intact and the second wound healed readily in seven weeks and three days, resulting in a neat linear scar.

At the second stage the dissection is carried up to the lower border of the healed wound. The resulting cavity is short for its depth and easy to keep packed, so that fecal contamination and unnecessary motion are minimized. Even under the best circumstances, however, the lower wound is more troublesome than the upper, since it lacks the splinting effect of underlying bony structures, is more vascular, and is subject to greater contamination.

After Care.—The original dressing is intended to minimize postoperative pain, bleeding and sepsis. The cavity is lined with sterile gauze soaked in thromboplastin, and then more gauze is introduced, soaked in a very mild antiseptic such as aqueous metaphen, mercurochrome (2 per cent), or hexylresorcinol until the cavity is snugly, but not tightly, packed; an analgesic paste such as butesin or nupercaine ointment is spread over the skin edges. A large sterile pad is then tightly held in place by two wide strips of adhesive plaster across the back.

The patient is allowed to walk home at once unless he has taken a general anesthetic, when he is kept in the hospital at least over night. He is warned to hold the lower edge of his dressing in against the base of his spine during defecation to avoid soiling. If comfortable, he may return to work the first day after operation.

The outer pad is changed as necessary, but the pack is not removed until

the fourth day. No coagulant is used at this or at subsequent dressings. We prefer not to use butesin ointment again after the first dressing since having seen two cases of butesin dermatitis apparently resulting. We have found liquid obtundia useful in subsequent dressings of painful wounds, though its odor is rather pronounced. After the second dressing, the wound is repacked as often as necessary until all necrotic tissue has disappeared, usually in about one week.

From the time the wound becomes clean until it has filled with granulation as much as it will, dressings may be done as far apart as twice a week. During this phase of healing our object is to do nothing to inhibit the growth of tissue cells, and if possible, to stimulate their growth. A balance must therefore be struck between too great ardor in the use of chemicals to destroy bacteria, (and at the same time tissue cells) and too great apathy towards harmful bacterial invasion of the wound. Only by personal supervision of the wound can such a balance be struck. We have found the use of zinc peroxide ointment in the depths of the wound helpful when we employed an active preparation, and apparently harmless when we used an inert one, in accordance with Meleney's[5] experience in other conditions. Care of the surrounding skin must not be neglected during this time. We allow several layers of adhesive plaster to accumulate before removing them, cleaning the skin with ether, painting it with tincture of benzoin, and starting a fresh accumulation of adhesive in a new place. At each dressing the skin about the wound is shaved and cleansed and covered with a thin protective coating of zinc oxide ointment.

In the present series we thought it necessary to trim away bits of doubtful-looking granulation tissue in 27 cases with scissors, in 33 cases with a sharp curette, and in four others with the cautery, sometimes with the help of novocain but more often not. We feel sure that a certain number of failures were prevented in this way.

Small plastic procedures were carried out before the wound was healed in nine cases. One was dissecting up a skin flap which had grown down to the wall of the wound because the packing had fallen out and remained out for a week. Four consisted of trimming off a raised skin edge which had become epithelialized on its under side. One of these edges was a corner formed by a transverse, accessory incision. Another procedure was the excision of a strip of skin between the incision and a lateral opening, which threatened to turn into a tubed graft. Three were skin incisions made parallel to the wound to hasten the final stage of scar formation by removing tension from scars which showed a tendency to longitudinal cracking.

As the granulating tissue approaches the level of the skin, new epithelium begins to grow across it from the skin edges. At this stage we change to a dressing designed to stimulate epithelial growth. A strip of sterile gauze soaked in thioglycerol is laid along the wound and thickly covered with zinc oxide ointment to keep the thioglycerol from soaking up into the dry part of the dressing. Although we have been unable to prove it quantitatively, our

clinical observations convince us that thioglycerol does promote epithelial growth.[6, 7]

Scar Formation.—In wounds which heal rapidly there appears to be a very perfect synchronization between the granulation and epithelial tissues, the former slowing down as it approaches a normal depth, the latter speeding up to form a level scar. In many wounds, however, due to the intervention of unfavorable factors, this synchronization is lacking. When the epithelialization is too slow, the granulation tissue may grow up beyond the normal level, forming a sort of coxcomb which has to be kept trimmed down until the epithelium can grow across. We have not seen this happen when thioglycerol was being used.

When the granulation is too slow, the epithelium begins growing down into a groove or cavity for which it will eventually form a lining. We no longer make any effort to prevent this development, as our follow-up examinations have shown that most of the resulting depressed scars tend in time to assume a normal level. When the groove into which epithelium is growing is very narrow, however, a surprising phenomenon is often observed. Instead of growing down the walls of the groove, the epithelial membrane will bridge straight across, leaving an empty space beneath its under surface. If this is not prevented, a false scar will be formed, which will later break down revealing one or more indolent granulating cavities of varying depths. About 20 per cent of our cases exhibited this phenomenon.

Where both granulation tissue and epithelium are too slow the wound is in danger of becoming essentially a chronic ulcer. After failing to stimulate tissue growth by various means, we have several times succeeded in obtaining a satisfactory scar by the use of pin-point skin grafts.

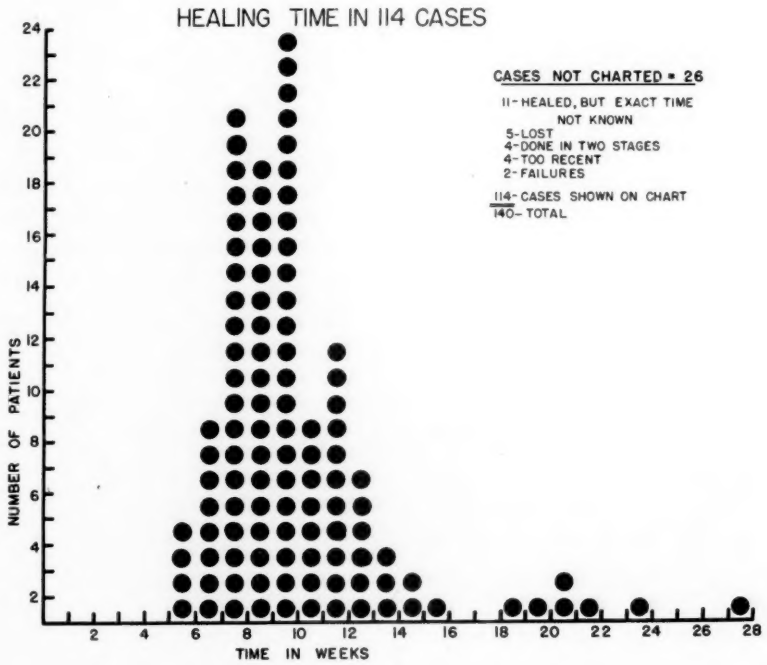
Healing Time of Wounds.—The time required for complete healing of the wound is accurately known in 114 cured cases (Table I).

It will be noted that the time is less than 14 weeks in 90 per cent of the cases, less than 12 weeks in 80 per cent, and less than ten weeks in 64 per cent, with a minimum of five weeks. The 10 per cent of patients whose wounds took longer than 14 weeks to heal present an interesting and complicated problem. After tabulating the size of the wounds, the healing time, and the sex of the patients, we are convinced that the sex and size of wound play no significant rôle in the rate of healing. We are unable to generalize about the rate of healing since we feel that all of the factors to be discussed under the head of complications must play significant parts.

Other things being equal, the comparative usefulness of a method must be judged: First, by whether it cures the patient; and second, by how long it takes to do it. A long healing time is obviously undesirable, and everything should be done to shorten it. We believe, however, that those patients who took so long to heal would probably not have been cured at all by other methods, for the factors which in other methods produce failures produce only delay in this. In other words, we believe that this method is fool-proof to a high degree.

COMPLICATIONS.—*Bleeding:* Postoperative bleeding, which was encountered eight times in our hospital series and twice in private practice, was never of serious consequence. It appeared to be due to one of two causes—imperfect hemostasis, with bleeding within 24 hours, or opening of a small vessel due to the separation of a slough between the sixth and eighteenth postoperative day. In three cases the bleeding took place within 24 hours; in the other seven it occurred 6, 7, 7, 7, 12, 18, 18 days after operation. The bleeding was stopped in five cases by cleaning out the clot and applying a fresh gauze pack. In the other five cases the bleeding vessel was seen and either crushed with a hemostat, cauterized, or ligated. The wounds healed

TABLE I



normally in all of these ten cases, and all ten patients are cured and symptom free.

We believe that better operative technic will reduce both types of bleeding to a minimum. During excision each small artery must be directly cauterized as it appears. The temptation is to ignore a little bleeder for the moment and defer the control of hemorrhage until the excision has been completed. This delay gives an occasional vessel an opportunity to retract and escape cauterization, only to reopen and bleed when the local adrenalin effect has worn off and the patient's blood pressure has become elevated by activity.

Excessive slough in the wound is always caused by trying to divide tissue when it is relaxed instead of held under tension. The cautery then boils and fries the tissue but cannot divide it cleanly. At the end of an operation, the

surgeon's left hand should be lame from holding the tissue forceps while his right hand should have exerted no force whatever in the manipulation of the cautery. Excessive slough not only predisposes to secondary hemorrhage, but causes pain and delays healing.

Pain.—The majority of the patients in this series required no sedation, lost no sleep, and experienced nothing other than soreness on sitting down during the first few days after operation. The discomfort was invariably greatest between the third and fifth day, as this is the time when the devitalized tissue is separating, and since the most severe pain has been noted in the cases of patients having the greatest amount of slough, we have concluded that the pain is caused chiefly by the exposure of sensory nerve filaments to the irritating influence of gauze, discharges, and air. Slough associated with skin or with sacral fascia causes the most pain because these structures are more sensitive than fat. Direct burns of the sacral fascia are capable of causing immediate pain.

Seven patients experienced severe pain, in four instances sufficient to bring them back to the Emergency Ward after operation. In one the pain began on the day of operation and reached its height on the fifth postoperative day. In the others the pain started about 48 hours after operation and increased up to the fifth day. They became comfortable between the seventh and fourteenth postoperative day. In two of these cases the excision had been performed by surgeons who were trying it for the first time, and in another a defective cautery was employed. The other three excisions were all very extensive, but should not, for that reason alone, have resulted in painful wounds. Six of the painful wounds healed in 7, 8, 10, 11, 11, and 15 weeks respectively. The seventh will be discussed in connection with abscess formation.

Fungus Infection.—Repeated clinical observations have led us to believe that many instances of delayed healing, and some failures, are caused by the spread of a fungus infection from the patient's perineum or feet to the pilonidal wound. This complication is characterized by arrested progress, tenderness or pain and sometimes itching, a disagreeable sour odor, and a white, moist, crumbling skin edge around the wound. It is relieved by the application of any simple fungicide, for example: Two parts each of acid salicyl. and sulphuris praec., and 30 parts of petrolat (M.G.H. ointment No. 20). This ointment may safely be used in either full or half strength. The condition has been observed in varying degrees in perhaps 30 cases in our present series. In retrospect we think it likely that many of our cases of prolonged healing were due to this unrecognized complication. Three striking cases are appended.

Case 1.—R. J., female, age 26, had a very small excision, and progress was excellent for six weeks, when the patient complained of soreness, itching, and sour smell which she had not noticed before. This increased for two weeks inspite of frequent dressings. At eight weeks the marginal skin looked boggy and white; the macerated epithelium could be scraped off, revealing a bluish, inert base. The resemblance to macerated skin between

the toes in epidermophytosis was so marked that we applied M.G.H. ointment No. 20 to the whole wound as an experiment. Within four days all her symptoms had ceased, and healing was complete two weeks later, ten weeks and four days after operation.

Case 2.—S. S., male, age 21, had a small excision, did well, and was almost healed at nine weeks. He then complained of itching, soreness and a sour smell. The wound had partially broken down with maceration of the skin edges and loss of new epithelium in spots. He got worse for two weeks. M.G.H. ointment No. 20½, was then started; the symptoms disappeared, and the wound was all healed 12 weeks and six days after operation.

Case 3.—P. S., male, age 22, had a small excision, with a resultant unusually clean wound and rapid healing up to 20 days. The skin edges then became macerated, granulation tissue edematous, progress stopped, and the patient complained of soreness. At four weeks a sour odor was present in the wound, and the patient complained of itching. M.G.H. ointment No. 20 was applied. At five weeks the symptoms had ceased, granulation tissue looked normal, a thin film of epithelium was growing out from the skin edges. The wound was completely healed at nine weeks.

Abscess Formation.—The convalescence of six patients was complicated by the formation of an abscess. All six operations had been performed under local anesthesia, but in only two cases could this have been a factor. In one an abscess appeared 4 cm. from the wound seven days after operation and required incision and drainage through the intact skin. It then subsided rapidly, and convalescence was uneventful. In the other, excision was carried out in spite of what appeared to be a pimple, but probably was a small furuncle, 1 cm. from the midline opposite the center of the incision. By the fourth day tenderness was marked and poultices were instituted. On the eighth day pain brought the patient to the Emergency Ward where, after 48 hours of poulticing, an abscess at the site of the pimple opened spontaneously and discharged one-half ounce of pus. An additional abscess appeared on either side of the incision, both of which required incision and drainage. Convalescence was uneventful.

We believe that novocain should not be injected through an infected area into a clean one which is not going to be given ample drainage by the impending excision. For this reason we limit ourselves to a midline, intradermal wheal of novocain along the line of incision, extending the anesthetic field by injection under the skin edge only so far as we expect to undercut it. We anesthetize the sensitive sacral fascia only under exceptional circumstances, and then with misgivings. Whenever it appears that extensive scarring will force us to violate these principles, we prefer to use a general anesthetic. General anesthesia was used in 14 out of 148 excisions.

In three cases an abscess appeared near to, but apparently unconnected with, the wound of excision from six to seven weeks after operation, and all required evacuation of the pus by incision and drainage. Better care of the skin might have prevented them. In none of the five cases mentioned did the abscess interfere with a successful outcome. The fifth case presented a complication which is fortunately rare. The patient was a stolid man, age 40, who had, at operation, "hair in the midline with scar and inflammatory tissue extending three inches into the buttock on the right." For four weeks after operation he was never free of pain. Rectal examination showed indura-

tion and tenderness in the right ischiorectal fossa, which on incision through the buttock, under general anesthesia, proved to be a large abscess communicating with the pilonidal wound. No opening within the anus or rectum could be found. The patient was immediately relieved of pain, and both his wounds healed uneventfully, but it is too soon to know whether he is cured. In retrospect it is obvious that a rectal examination should have been made sooner, as the promptness of his pain suggests that even at the time of operation extension into the ischiorectal tissues may have been present.

Defects in the Scar After Healing.—*Superficial Maceration:* In five cases small areas of superficial maceration appeared in the scar from 12 days to one year after healing. In each instance there was evidence of poor hygiene and lack of care of the scar. When this was remedied the macerations disappeared in from two to seven days. All patients, on discharge, are instructed to wash and powder the scar daily, and protect it by a small strip of absorbent cotton held in place by the underwear. After several month the scar is usually tough enough to take care of itself.

Longitudinal Cracking.—Of this there were four instances, occurring from 17 days to one and one-half years after healing, no crack exceeding 1.5 cm. in length, and none taking longer than eight days to heal. They all were due to some sort of physical violence, such as a fall in the sitting position. A fifth patient scratched his wound open in his sleep, because of intolerable itching during hot weather, seven days after it had healed. Healing was again complete in three weeks.

Bridging.—In about 20 per cent of the wounds the last epithelium to form showed a tendency to bridge across narrow defects instead of growing down into them. In one case we neglected to cut back this bridging epithelial membrane, so that seven days after healing was apparently complete a pinhole-sized opening was discovered in the scar. A probe introduced through it entered a granulating cavity 1 cm. deep and 0.5 cm. wide, containing no evidence of a sinus in its floor or walls. After a few days of packing the cavity healed up from the bottom to an intact scar.

Epithelial Pouches.—In three cases a pouch was formed by the downgrowth of epithelium into a fairly deep granulating cavity. One was 2 cm. deep with an intact lining of dry skin, and caused no symptoms. The two others became inflamed three and one-half and seven months after healing, apparently due to poor hygiene, and presented a picture similar to omphalitis. Both required small plastic procedures to enable the patients to keep their scars clean. Pouch formation is merely an exaggerated form of the depressed scar, which usually tends to flatten out spontaneously as the scar tissue becomes soft.

Superfluous Skin.—Two patients developed completely epithelialized bridges of skin beneath which a finger in one case and a probe in the other could be passed. The larger resulted from the surgeon's unwise employment of a second incision parallel to the midline incision when he found himself unable to reach a lateral extension of scar tissue from the midline. In the process of

healing, the intervening strip of skin tubed itself, the new epithelium growing completely across underneath it. In the second case the skin tubed itself between the midline incision and an unusually large lateral sinus opening. This development was prevented in several other cases by sacrificing such narrow bands of skin. Although such bizarre skin formations may cause no symptoms, they delay healing and should be avoided. When scar tissue cannot be reached by undercutting from the midline, it should be exposed by a second incision at right angles but never parallel to the midline incision.

Late Abscess Formation.—Our most troublesome case was one in which an indolent abscess formed in the lower end of the scar one month after the wound had become healed, and continued to drain for two months before the patient returned to the clinic. Believing that this abscess cavity, even though it contained no hair or epithelium, was capable of producing a persistent sinus, we excised it with the cautery. The wound then healed after 27 weeks of packing, and has remained healed for several months. Fungus infection appeared to be a factor in the unusually slow healing, and may have been concerned also in the formation of the abscess.

Residual Symptoms.—Of the patients known to be cured for at least one year, 70 (49 male, 21 female) have been questioned about posthealing symptoms. Fifty (71 per cent) have no symptoms and are never conscious of their wound. Eighteen (26 per cent) have, or have had, symptoms of minimal magnitude. Two (3 per cent) have residual pain. Thus, 97 per cent of this unselected group have complete or almost complete freedom from residual symptoms.

The minimal symptoms fall into two general classes: Those associated with long sitting on a soft substance, *i.e.*, pressure; and those due to other activities or conditions, or having no known association with anything.

Of the 11 patients in the first class, five described a "faint dull pain" on rising. In one case this ceased after one year, in another after one and one-half years. Three patients still have it, but one only after a long automobile drive. Two patients described "discomfort," and one each "soreness," "heavy feeling," "numb feeling," and "pulling sensation."

Of the seven patients in the second class, two described dull pain off and on during damp weather, one had a single twinge of pain two years after healing, one has "occasional slight momentary pain," one had "slight pain" for three weeks, one has itching in warm weather, and one has a "tight sensation on stooping over sometimes."

The two patients with more than minimal symptoms are of interest from a diagnostic as well as a therapeutic point of view. Both were females, and both had smaller than average wounds.

Case 1.—A. B., a Negress, for one and one-half years after healing, had been suffering from what she described as "an ache like a toothache" which prevented her lying on her back or slouching in a chair. It always came on after long sitting and occasionally at other times. She said the pain never radiated. Examination showed an intact, soft, linear

scar 5 cm. long. There was slight tenderness over the sacrococcygeal joint, but no pain on manipulation of the coccyx through the rectal wall.

Case 2.—C. D., was symptom-free for six months after healing. She then started doing gymnastics and began to notice that it caused pain in the wound, as did playing tennis during the summer, until by fall, sitting down for long periods also caused an aching pain, described as a dull ache at the lower end of the sacrum and sometimes radiating "up the spine." Examination showed an intact, soft, freely movable, almost invisible linear scar, with tenderness confined to the sacrococcygeal joint, but no pain on moving the coccyx from behind or through the rectum.

In reviewing the phenomena of residual symptoms, we find nothing that throws any direct light on its mechanism. Of the 20 patients concerned, 18 had wounds of average or smaller than average size. There is nothing about the rate of healing or the resultant scars which singles these individuals out from the rest. The only finding which may be of any significance is the sex incidence of those suffering from residual symptoms. We found an equal number of males and females, instead of the 3:1 ratio characteristic of the series as a whole.

If this sex incidence of those having residual symptoms is more than a coincidence, it suggests that the anatomic differences in the male and female pelvis may be concerned. Duncan[7] has pointed out that these differences expose the female coccyx to more trauma than that of the male, which is relatively well protected by its more pronounced forward curve. Other things being equal, this difference may account for the high relative incidence of painful coccyx in females (97 per cent) and the high incidence of pilonidal sinus in males (66 to 80 per cent), since the trauma which plays a rôle in the etiology of pilonidal sinus in males is in females absorbed by the more prominent coccyx. After operation for pilonidal sinus, however, the situation becomes altered, for in both sexes there is now scar tissue overlying the coccyx, but in the female it is more exposed to direct trauma than in the male. Assuming that trauma to scar tissue could cause such symptoms as we have observed, the sex distribution might be accounted for on this basis. If no other factors than scar tissue and trauma be responsible, however, it is odd we do not find residual symptoms in all cases, since these same factors must be present in all.

Our experience leads us to the conclusion that a certain incidence of such residual symptoms as we have described is unavoidable following surgery in the sacrococcygeal region, and that if they are not mentioned by other observers it does not mean they are not there, but that they have not been looked for.

FAILURES.—In the case of two patients who were thought to be cured, a pin-hole opening was later discovered below the bottom of the scar. One was found on routine follow-up examination one year and four months after the patient's last visit, at which his wound was not entirely healed. It was a tiny opening resembling an enlarged pore 2 cm. below the lower end of a well healed scar, and from it protruded two or three hairs. It had not caused symptoms, and the patient was not conscious of its presence. The other pa-

tient was examined 13 months after his last visit and found to have a pin-hole opening with a protruding hair just at the lower end of a well healed scar. It had drained once in a while, but he did nothing about it as it "didn't bother him much."

These small openings were missed, presumably because at the time of operation they contained no hair. Even had they been seen, as with more care they would have been, it is probable that the same operative procedure would still have been carried out, but with the expectation of a second stage if developments should prove it to be necessary. That is what will be done now with every prospect of cure, but honesty demands that these two cases be rated as failures at the present time.

SUMMARY.—A series of 140 patients with a 3:1 male to female distribution, all treated by cautery excision and open packing, is described.

The rationale of this method of treatment is discussed.

The technic of cautery excision and care of the wound is reviewed in the light of the authors' experience, with particular reference to complications.

The results of treatment are presented in some detail.

CONCLUSIONS

Since, in a personal experience with some 400 cases of pilonidal sinus we have not yet seen one communicating with the neural canal, we conclude that it is a rare occurrence. Roentgenographic examination with iodized oil injection is superfluous except in markedly atypical cases.

We have never seen cancer develop in a pilonidal sinus.

Under ideal conditions for visualizing the gross pathology of pilonidal sinuses (*i.e.*, cautery excision) we have seen none extending into the sacral hiatus, sacrococcygeal joint, or beneath the sacral aponeurosis.

The two essentials for cure are: (1) Removal of diseased tissue; and (2) solid healing of the wound.

Though the technic of removing diseased tissue is unimportant compared with the subsequent treatment of the wound, we see no advantage in performing its excision by a difficult procedure when a simple method is available (*i.e.*, cautery excision).

Because of the unavoidable obstacles to sound healing by first intention, in pilonidal wounds, we believe that open packing and healing by second intention will produce the highest percentage of cures in unselected cases.

In selected cases it may still be desirable to incur the greater risk of failure associated with primary closure for the sake of shortening the treatment time in those which succeed.

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TREATMENT OF FRACTURES OF THE SHAFT OF THE FEMUR*

AN ANALYSIS OF ONE HUNDRED TWENTY CASES

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FRACTURE of the shaft of the femur is a common, severe injury, frequently resulting in death, permanent disability or prolonged invalidism. Any information which may aid in improving these results should be made available. We can determine whether certain types of treatment should be continued only by knowing what can be expected of them. The evaluation of a method depends upon a comparison of an analysis of a group of cases so treated with analyses of other methods. We have analyzed 120 consecutive cases of recent fracture of the shaft of the femur treated by the staff of the Beekman Street Hospital, from January, 1924, to March, 1932. It seems worth while to make these observations available in order that other methods, as they have been or are devised, may be compared with them, in order to determine their relative value.

Before the War of 1914–1918, the usual handling of these patients consisted of reduction under anesthesia followed by immobilization by plaster encasement. Sporadic attempts had been made to introduce traction in the form of Buck's extension or insertion of a Steinman pin and also open reduction with internal fixation. None of these methods made much progress, and reduction and plaster encasement continued to be the routine treatment. This was difficult to carry out in compound fractures and, in all types, resulted in shortened limbs, poor weight bearing axes and loss of joint function with appalling regularity.

At the commencement of the war, application of a plaster encasement or internal fixation of fragments was the common procedure in compound fractures of the femur. It soon became evident that the results of either method were too unsatisfactory to continue their use. Traction methods became popular, at first by pull on the skin and later by pull directly upon the distal fragment of bone.

On returning to civil practice many surgeons reverted to the plaster encasement of both simple and compound fractures, but the majority of those with a particular interest in fractures continued to use some form of traction. The latter believed that the original length, proper axis and complete function were to be desired in every case, and that these results were difficult to obtain following immobilization by a plaster bandage.

Beekman Street Hospital was reorganized in 1924, and the senior mem-

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bers of the new surgical staff had all had war experience. They were uniformly in favor of some form of traction and against the use of a plaster encasement unless the type of fracture was such that slipping after reduction seemed unlikely. The institution is a general hospital of 100 beds with a single operating room. The house staff has a rotating service. It receives many infected cases and is in a district where gas bacillus infection is common. These are the chief reasons why it was thought that open operative reduction should not be employed by the staff unless other methods had proved unsatisfactory. That is, open reduction was not performed as a primary treatment of choice in fractures of the shaft of the femur. However, for several years, any open operative bone work has been in charge of one surgeon only. This arrangement was begun after the period which this report summarizes.

The patients to be considered have been treated by some ten surgeons, although each case has had the advantage of general staff discussions of the details of the treatment. We believe that the proper transportation of the patient to, and within, the hospital is a major factor in treatment.[2,3] The routine use of fixed traction on our ambulances was insisted upon during the latter half of the period considered.

There were 17 early deaths in this series. Fourteen had multiple, severe injuries of which the fracture of the femur formed only a part. One died within 48 hours of alcoholism and delirium tremens. One patient had fractures of both femora, one of which was compound. A débridement of this was effected and adhesive plaster traction instituted. He died within 24 hours from a cerebral embolism. The other instance of early death occurred in a case of severe compound comminuted fracture of the lower third of the shaft with severe hemorrhage. His condition was such that only packing of the wound was feasible the first day. A débridement was effected on the second day, and an amputation through the site of fracture on the fourth day, on account of gangrene of the leg from disturbance of the blood supply. The patient died on the sixth day. The time of death in these 17 cases is shown in Table I.

TABLE I

TIME OF DEATH IN 17 EARLY MORTALITIES

Time Elapsed	No. of Cases	
Within 2 hours	6	
Between 2 and 12 hours	6	(Three of these were compound; skeletal traction had been instituted in one)
Between 12 and 24 hours	2	
During the second day	1	(Compound; skeletal traction had been instituted)
During the fifth day	1	(In skeletal traction)
During the sixth day	1	(Amputation)

In addition to the death following amputation, two other patients had amputations and survived. One was a child, age nine, who had an extensively

comminuted compound fracture of the middle third of the shaft with maceration of the soft parts of the leg. A transfusion was given shortly after admission followed within 24 hours by a second transfusion and a disarticulation through the knee. On the twenty-third day an elective amputation was performed through the middle of the thigh. The other patient had a closed fracture of the middle third with extensive hemorrhage into the thigh. By the fifth day he had developed a deep cellulitis of the thigh. In spite of surgical treatment, the cellulitis continued to extend and an amputation, through the upper third of the thigh, was performed on the nineteenth day.

There remain 101 patients available for more detailed analysis of their treatment. Since the problem varies somewhat in different regions, the series has been divided into three groups: the upper, middle, and lower thirds.

Upper Third.—This group does not include fractures of the greater or lesser trochanter nor intertrochanteric fractures, except when these accompany, as relatively unimportant features, fractures of the upper third of the femur in the same patient.

There were 17 patients in the group, 14 males and 3 females. Their ages ranged from 5 to 15 years, 2; 16 to 29, 4; 30 to 39, 4; 40 to 49, 1; 50 to 59, 2; 60 to 69, 4. There were no compound fractures. Four were comminuted.

The causes varied from slipping on a banana peel or a rug to falling six stories, four stories and from an elevated railroad platform to the street. Only four were the result of motor accidents. In 11 instances the trauma seemed severe and in six relatively slight. The appearance of the roentgenogram did not vary greatly, irrespective of the severity of the violence. Two persons were struck locally with heavy objects and three others believed that they had sustained a direct blow on the upper thigh in falling short distances. Whether the fracture was due to direct or indirect violence in the others was undetermined.

In one instance the lesser trochanter formed a part of the long distal fragment; in one case it was broken off separately; in another both the greater and lesser trochanters were broken off separately; and in a fourth the greater trochanter was separated. In one patient there was a long oblique fracture commencing just below the lesser trochanter and in addition a transverse fracture in the middle third of the femur. This patient had other serious injuries in addition which increased his disability: namely, fracture of the bodies of the second and third lumbar vertebrae, fracture of the patella and calcaneus on the same side as the fractured femur, and fracture of the tarsal scaphoid and second metatarsal on the opposite side. Three other patients had complicating injuries as follows: Fractures of the pelvis; laceration of the brain and fracture of the olecranon process and multiple fractures of the pelvis; fracture of two ribs.

The typical displacements in this fracture are abduction, flexion and external rotation of the proximal fragment, as a result of the action of the muscles attached to the trochanters and the intertrochanteric line. In most

of these patients the first roentgenologic examination was made with the extremity in emergency traction. In spite of this traction, nine of the initial roentgenograms showed, to some extent, the typical deformity, which makes this type so difficult to treat.

The first definitive treatment in the hospital was adhesive plaster traction with Thomas splint suspension in ten patients, ice tongs in six, and overhead traction to both legs in a child, age five. The adhesive plaster traction treatment was unsatisfactory in four instances. In one of these a closed reduction, under general anesthesia, was attempted on the fifth day and a plaster spica applied; an open reduction with Lane plating was effected and a plaster spica again applied on the eleventh day because the reduction was not maintained. In two other patients ice tongs were substituted, and in one a Steinman pin was introduced through the distal portion of the femur. That is, the final treatment was skeletal traction in nine instances, adhesive plaster traction in six, open reduction in one and overhead traction in one.

In using adhesive plaster, the first weight applied varied between 12 and 40 pounds, being 15 pounds in five cases; the permanent weight was usually 15 pounds. With skeletal traction the initial weight applied varied between 20 and 35 pounds, averaging 27½ pounds. It is evident that, in general, adhesive plaster was employed where it was thought less traction was necessary, since the effective pull averaged only slightly more than one-third as much. In addition six of these ten cases occurred during the first three years of the period reported, at which time we employed skeletal traction less frequently. In five instances the adhesive traction treatment was commenced immediately after admission and was satisfactory four times. It was begun the day after admission three times, twice unsatisfactorily, and not until the second day twice, being unsatisfactory once.

When roentgenlogic examination demonstrated that the desired position had been attained, 15 pounds was the usual amount of skeletal traction maintained. Skeletal traction was not removed until the one hundred ninth day in the patient with fractures of both the upper and middle thirds of the femur. One patient died of embolism on the seventeenth day. Omitting these two, the tongs were removed in the remaining seven patients between the twentieth and forty-eighth days, averaging 38 days. In all but one instance adhesive plaster traction was substituted, usually with 15 pounds' weight. Omitting the same two patients as above and also the case of open reduction, in the remaining 14 all traction was removed at from 38 to 83 days, averaging 62 days. One patient, from whom all traction was removed on the sixtieth day, refractured on the seventy-first day, when commencing to use a walking caliper splint. He had an open reduction performed on the seventy-sixth day, with application of a Lane plate, followed by traction suspension with a weight of ten pounds attached to adhesive plaster.

We know that nine patients obtained solid union and one died. For various reasons the remaining seven could not be followed, but the condition when last seen gave no reason to suspect that they would not obtain union.

The time of first appearance of any callus, as demonstrated roentgenologically, is mentioned on 14 charts. This varied between 28 and 65 days and averaged 42 days. Since the usual period between roentgenologic examinations at this stage in treatment was two to three weeks, this figure cannot represent, accurately, the first appearance of callus. The precise time of appearance of firm union or solid union is not mentioned on enough charts to make this figure of any value.

Ice tongs or a Steinman pin was employed in nine instances. One patient died of pulmonary embolism on the seventeenth day, with no infection of the tong wounds. In three other patients the wounds remained clean. Five became infected. One was transferred to another hospital 11 days after removal of the Steinman pin, and the result is not known. One still had a purulent discharge 30 days after removal of the tongs, but this condition is not mentioned in the notes for the following three weeks, after which he was lost track of. Another still had a discharging wound four and one-half months after removal, when he disappeared. One drained for at least six months, but we know that nine months later he had no trouble for many months. Another drained for eight months after removal, but following the extrusion of a small sequestrum the wound healed and remained so. Some of these cases did not drain continuously, but would apparently heal, only to open again after a few weeks.

One patient died during treatment, and we have the late result noted in only six others. Four were A_4, E_4, F_4 (Massachusetts General Hospital rating). One was A_4, $E_?$, F_3. We gave him F_3, because his knee motion was from 180 to 80 degrees, and he had persistent edema of the leg, one year and four months after injury. He sustained a refracture through his callus on the seventy-first day, and had an open reduction on the seventy-sixth day. Another patient was given A_4, E_0, F_3. He had not returned to work at the end of three years on account of the fracture of two lumbar vertebrae. He was given F_3, because he had 90 degrees flexion at the knee.

We do not believe that these patients are usually satisfactorily treated in plaster encasements. While we used adhesive plaster traction in six of our early cases with fairly satisfactory results, it failed in four other cases, and we do not believe it should be used generally. Overhead, bilateral adhesive traction is satisfactory in patients under five years of age. Skeletal traction gives excellent anatomic and functional results, but an incidence of infection of the wounds of 55 per cent is a severe indictment of the procedure or of our method of using it. At present we would employ the Kirschner wire for skeletal traction, instead of ice tongs or a Steinman pin. It has been our experience that superficial soft part infections are even more common with the use of Kirschner wire than with the tongs or pin, but that prolonged discharge after removal of the wire, which we consider an evidence of bone involvement, is rare.

Middle Third.—There were 74 patients in this group, 64 males and 10 females. Their ages were as follows: up to 16 years, 42; 16 to 29, 9; 30 to 39,

6; 40 to 49, 8; 50 to 59, 6; 60 to 69, 3. Twenty-five were the result of motor vehicle accidents, and the next most common cause was falling from a height. Two were compound and two were comminuted fractures. The fact that there were 42 children in this group is important in the incidence of this fracture, for the hospital covers a large business, but small residential, district, in which there are comparatively few children. We shall discuss the children's group separately. Fifteen of these were not over five years of age.

The roentgenologic reports state that there were 11 transverse, ten oblique and four spiral fractures. Overriding is mentioned as being present in 19 instances. Some of these occurred in the early years before we employed traction splints on the ambulance, and some were brought in by conveyance other than an ambulance. Overriding is rarely seen when a fixed traction splint has been applied before admission. Without it, marked overriding is characteristic in middle third fractures.

Eleven patients had additional injuries as follows: Concussion of the brain, three; fracture of the skull, three; humerus, two; nose, one; jaw, one; and patella, one.

The first definitive treatment in the hospital for the 15 patients under six years of age was overhead adhesive traction to both lower extremities in ten instances (Bryant traction). This was continued until union was obtained in every case. We do not believe overhead traction to one extremity only should ever be employed. Closed reduction and a plaster spica was employed three times. It was satisfactory in only one instance. In another case it was employed three times before being satisfactory. In a third patient, after the reduction was unsatisfactory once, adhesive plaster traction with a Thomas splint was successful. In two cases this latter treatment was employed primarily, once with a good result and once followed by successful closed reduction and application of a plaster spica. That is, the final treatment was overhead traction in ten, adhesive plaster traction in two, closed reduction and application of a plaster spica in three. Bryant traction is an excellent method for patients under six years of age.

For the 27 patients between the ages of six and 15, the Thomas splint with adhesive plaster traction was employed as the first treatment in 21 instances, 17 times satisfactorily. Two of the unsatisfactory cases had a secondary closed reduction under anesthesia with application of plaster spicas. A third patient had a Steinman pin inserted through the crest of the tibia, and the fourth unsatisfactory reduction had an open reduction with kangaroo tendon suture, interposed muscle being found. Four patients had closed reductions under anesthesia with plaster spica application as their primary treatment. This was successful three times. In the other case it was repeated, and a Thomas splint with adhesive plaster traction was tried, and finally an open reduction was performed without fixation. The two compound fractures had a débridement, one having a plaster spica applied followed by open reduction and wiring on the twenty-sixth day for malposition, and the other the insertion of a Steinman pin through the crest of the tibia. That is, the

final treatment was adhesive plaster traction in 17, closed reduction and the application of a plaster spica in five, open reduction in three, and skeletal traction in two. We believe that closed reduction with the application of a plaster spica is not usually a satisfactory treatment. In all adhesive plaster methods we employ manipulation in bed, under anesthesia, in addition, if satisfactory position is not obtained promptly. Traction is immediately re-instituted.

With Bryant traction the weight is the same on each leg and should be sufficient to just keep the buttocks from touching the bed. We do not fix the trunk to the mattress or to a Bradford frame. The weight varies between four and eight pounds. Fifteen pounds was an average weight for adhesive traction in a Thomas splint. It differs, naturally, with the size and weight of the patient. Irrespective of the site of fracture, each adhesive plaster strip should extend as high on the thigh as possible and should be in one strip, and not divided to allow knee movement. To attempt the latter loses too much surface to which adhesive plaster can be attached. The final weight for skeletal traction was seven to ten pounds.

The ten patients treated by Bryant traction had no form of immobilization used later. The time of removal of traction varied from 37 to 56 days, averaging 45 days. Several of the patients treated in Thomas splints, with adhesive plaster traction, had the traction removed when union was firm and plaster spicas applied. In 11 instances adhesive plaster traction constituted the entire treatment. This was left on for periods varying from 23 to 62 days, averaging 47 days. One Steinman pin was removed after 65 days with no further immobilization, and one after 33 days, being followed by the application of a plaster spica. Seventeen patients had plaster spicas applied as their final treatment. This includes those originally treated by reduction and plaster encasement, as well as by adhesive plaster traction, skeletal traction and open reduction. The time of removal varied from 35 to 169 days after injury, with an average of 78 days. Of the older patients in the children's group, several used walking calipers after immobilization was discontinued.

In the group under six years of age the first roentgenogram showing callus is recorded 12 times. Roentgenograms are not taken as frequently in Bryant traction patients as in others, and those showing callus are usually the first roentgenograms taken after the immediate postreduction film. The time of the appearance of callus varied between 14 and 42 days, averaging 24 days. But in eight of the 12 patients it was noted between the fourteenth and nineteenth days, so that we may assume that at this age, callus is usually visible by the end of the second week. In the older children's group we have reports on callus formation in 20 instances. Because of the type of treatment, roentgenograms were taken at more frequent intervals. The time of appearance of callus was noted between the eleventh and thirty-sixth days, averaging 22 days.

In the group under six years of age, the time of appearance of firm union was noted in eight cases, and varied between 22 and 48 days, averaging 37

days. In the group between the ages of six and 15 it was noted in 15 instances, varying between 15 and 84 days, averaging 41 days.

Of the entire 42 children, four were lost before solid union was noted. There was no reason to expect delayed or nonunion in any of the latter. We know that solid union was obtained in all the others. One patient fell out of a chair on the fifty-sixth day and refractured the femur; union, however, became solid after 78 days more. One compound fracture, which had had an open reduction followed by osteomyelitis, did not become solid for one year. Omitting these patients, the time of solid union was noted in nine of the group under six years of age, varying between 25 and 74 days and averaging 54 days. In the group between six and 15 it was noted in 14 instances, varying between 38 and 106 days, averaging 72 days.

Of the two Steinman pin cases, the wounds in one remained clean, and in the other they became infected and discharged for four months after removal, before remaining healed. The two closed fractures which had open reductions healed without infection. The compound fracture with open reduction became infected and had delayed union.

Of the patients treated in Bryant traction, four showed no shortening, four, one-half inch and in two there was no record. Late roentgenograms, while still in the hospital, showed good axes in five, posterior angulation in two, anterior and lateral angulation in one, not reported in two. Late follow-up showed F_4 in six and no follow-up in four.

Some shortening was present at the end of treatment in 64 per cent of these patients. These are not satisfactory results. The two patients treated in skeletal traction had no shortening. Skin traction resulted in 62 per cent, and reduction and plaster encasement in 85 per cent shortening. It suggests that immobilization in plaster of paris is not, in general, a rational treatment. Skin traction in this series gave indifferent results.

TABLE II

AMOUNT OF SHORTENING IN CHILDREN BETWEEN THE AGES OF SIX AND 15

Amount of Shortening	Immobilization By			
	Adhesive Traction	Plaster Spica	Steinman Pin Traction	Open Operation
None	6	1	2	—
½ inch	4	3	—	—
¾ inch	2	—	—	1
1 inch	1	2	—	—
1½ inches	—	1	—	—
2 inches	1*	—	—	1
Not reported	1	—	—	1

* This patient signed a release on the thirty-seventh day and was not seen again until the ninety-second day, when a two-inch shortening was present. There had been ¼ inch shortening at time of discharge.

It is true that many of these extremities regain their normal length with growth, but we cannot consider efficient a treatment, which attempts to obtain

end-to-end apposition, if shortening results in the majority of cases. We have felt that this group of children, from six to 15 years of age, was the most difficult in which to obtain a satisfactory anatomic result in the shaft of the femur. We believed that it was inadvisable to use a Steinman pin or ice tongs in the femur on account of possible disturbance of the growth, unless this was absolutely necessary. We do not believe that this objection obtains for Kirschner wire through the distal end of the shaft proximal to the epiphyseal line.

Late roentgenologic examination, while in the hospital, showed good axes in ten, posterior angulations in six, and various other types of angulation in six. These angulations were usually slight. The result was F_4 in 17 patients, F_3, in one treated by a plaster encasement, with a restriction of knee motion still present at the end of one year, and F_2, in the patient with open reduction followed by osteomyelitis.

There were 32 patients over 15 years of age with fractures of the middle third of the shaft. There were no compound and 12 comminuted fractures in this group. Nine were apparently injured by direct violence, *i.e.*, some heavy object struck the thigh, 13 as the result of motor accidents, five by falls from a height and the remainder from miscellaneous causes. Two fractures were definitely spiral. Oblique fractures were about twice as frequent as transverse fractures. Nine cases were complicated by other fractures (Table III).

TABLE III

CONCOMITANT FRACTURES IN NINE INSTANCES IN PATIENTS OVER 15 YEARS OF AGE

Case 1.—Acromion process of scapula.
Case 2.—Rib.
Case 3.—Pelvis.
Case 4.—Upper extremity of femur, through base of neck, same side.
Case 5.—Upper extremity of tibia into knee joint, same side.
Case 6.—Shaft of tibia and fibula, middle third, and fifth metatarsal, same side.
Case 7.—External cuneiform and fifth metatarsal, same side.
Case 8.—Base of skull, mandible, patella and os calcis, opposite side.
Case 9.—Dislocated shoulder, supracondylar fracture of femur, opposite side; both patellae, both fibulae, upper third of shaft.

The first definitive treatment was with ice tongs in 24 instances, Steinman pin in the lower end of the femur in one, and in the upper end of the tibia in one, adhesive plaster traction in six. Plaster of paris encasement was not employed in any case as the initial treatment. We believe that satisfactory reduction cannot usually be maintained following manipulation and, even if it is primarily good, the danger of slipping later within the plaster encasement is too great. Adhesive plaster traction was satisfactory in three and unsatisfactory in three instances. Tongs were substituted in two of these cases. The third refused skeletal traction and was reduced and immobilized in a plaster encasement. The final treatment was skeletal traction in 28 instances, adhesive plaster traction in three, reduction and plaster encasement in one.

The initial weight applied with skeletal traction varied from 15 to 40 pounds as follows: Fifteen, twice; 20, 5; 25, 5; 30, 4; 35, 8; 40, 1; not recorded, 3. The greater weights were kept on for periods varying from two hours to two days and then reduced. Fifteen pounds was the usual weight employed for continued extension. Except for one patient, from whom the tongs slipped on the eighteenth day and adhesive plaster traction was substituted, skeletal traction was removed at periods varying from 30 to 92 days and averaging 53 days. Plaster of paris encasement was applied on removal of the traction in two instances, adhesive plaster traction in 24, and no further immobilization twice.

The wounds from skeletal traction remained clean in 14 and became infected in 14 instances. Discharge from tong wounds at any time, no matter how superficial, is classed as infection. In ten instances the condition was evidently a soft part infection only, as it cleared up promptly after the tongs were removed and remained healed. In four instances we believe there was bone infection as a result of the insertion of tongs. In one case a tong wound did not remain healed until one year after their removal, in another not until 20 months later. One was unhealed when last seen, two months after removing the tongs, and another 31 months later. This patient had been in another hospital twice for treatment of the tong wounds. This is an incidence of 14 per cent of bone infections from tongs, prolonging the period of treatment in each instance.

In the 24 skeletal traction patients to whom adhesive plaster traction was applied after its removal, the time at which all traction was removed is not charted in one case; in the remaining 23 it varied between 44 and 118 days, and averaged 82 days. In the three cases treated entirely with adhesive plaster traction, all traction was removed at 31, 57 and 165 days. Almost every patient used a hinged walking caliper after traction was removed. One patient had a refracture through the same site on the ninety-ninth day and another had a bone transplant from the tibia on the seventy-eighth day because of delayed union

We know that 29 of these patients obtained solid union, although the time of its earliest appearance is not stated on enough charts to make it informative. One patient was discharged in a plaster encasement after 29 days and lost track of. One was not seen after the sixty-third day when he was using crutches without weight bearing. He was said to have had firm union on the fifty-seventh day. The remaining patient was discharged on the sixty-sixth day and lost track of. There was no reason to expect a nonunion in any of these three patients from their progress up to the time of their discharge, or as a result of the position of the fragments.

The time of appearance of callus in the roentgenogram is mentioned on 28 charts, and averaged 36 days. The earliest was 15 and the latest 66. We believe that the average figure is materially less than 36 days since, after the position and weights used had reached a stable point, roentgenograms were not always taken frequently. In nine instances callus was visible in less than four

weeks, and we believe this probably approaches more nearly the correct figure. The time of appearance of firm union is mentioned on 25 charts, varying between 23 and 88 days, averaging 54 days.

The time at which the patient was allowed to walk wearing a walking caliper splint is mentioned on 23 charts. One was at 155 days in a case which refractured at 99 days, another was at 159 days in a patient in whom a bone graft was inserted at 78 days and a third at 193 days in a patient who was treated, from the start, by adhesive plaster traction and who had delayed union. The remaining 20 commenced walking at periods varying from 70 to 162 days, averaging 103 days.

The time at which free weight bearing without caliper support was begun is mentioned on 15 charts. One patient commenced walking at four months, against advice, and obtained an excellent result. In another patient no caliper was used, weight bearing was commenced at five and one-half months and a refracture occurred at nine months. Omitting these two, free weight bearing was allowed at times varying from seven to 13 months, averaging ten months.

Our knowledge of exact late results is limited. Most of these patients were compensation cases who were cared for by the insurance companies after discharge. We know that in some instances the patient was told that if he returned to our Follow-up Clinic he would be cut off compensation. If a patient usually is not walking without his brace before ten months, the final functional result will not be obtained before one and one-half to two years. We were not able to follow many of our patients for this length of time. Attempts to locate them later were rarely successful, as they could not be found, having changed their residences and left no forwarding addresses.

We have a record of shortening in three patients only, three-quarters, one, and one and one-quarter inches, respectively. Ten patients we know had an A_4, result. One was given A_3, on account of abduction of the upper fragment resulting in an external angulation. In the remaining 18 we have no anatomic result except that which was inferred from their last roentgenogram while under treatment.

Complete function at the knee joint is recorded in six cases. Other functional results are shown in Table IV.

Late function of the knee joint was not recorded in the remaining 18 patients.

We believe that skeletal traction is a satisfactory method of treatment for fracture of the middle third of the shaft of the femur. At present we would use Kirschner wire through the lower end of the femur for this purpose instead of ice tongs or a Steinman pin. Manipulation under anesthesia followed by plaster encasement is rarely a satisfactory method of treatment. Adhesive plaster traction with a Thomas splint is usually ineffective.

Lower Third.—These are placed in a separate group, because of the usual posterior angulation of the distal fragment, requiring different management. Fractures of the condyles are not included.

There were ten patients in this group, nine males and one female. Their ages were: Up to 15 years, 4; 15 to 29, 3; 40 to 50, 1; 50 to 60, 1; 60 to 69, 1. Two were compound, two comminuted and one greenstick. Five patients were struck by automobiles and three fell from a height. In one instance the shaft was driven down between the fragments, splitting them entirely apart. One patient had, in addition, a fracture of the clavicle and five ribs and another gunshot wounds of the foot and forearm.

In all except the greenstick fracture the typical tilting backward of the proximal end of the distal fragment was present. In only three of these nine

TABLE IV

At six months	
	180 degrees to 90 degrees
	180 degrees to 75 degrees
	175 degrees to 90 degrees
At nine months	
	180 degrees to 75 degrees
At ten months	
	170 degrees to 105 degrees
At eleven months	
	180 degrees to 140 degrees
At eighteen months	
	180 degrees to 90 degrees
At two years	
	180 degrees to 60 degrees

patients was it believed that this angulation was completely reduced, although in all the others it was so markedly improved that a good weight bearing axis was to be expected. The original angulation was as much as 45 degrees in some instances.

As first definitive hospital treatment, three children had reduction followed by application of a plaster spica; five had adhesive plaster traction with a Thomas splint suspension, the weight varying between 18 and 30 pounds; one, tongs in the femur (30 lbs.); and one a Steinman pin in the tubercle of the tibia (20 lbs.). No open operations were performed primarily. The adhesive plaster traction treatment was unsatisfactory twice and later Steinman pins were inserted in the tubercle of the tibia with 30 and 35 pounds weight applied. Skeletal traction was, therefore, used four times. This was removed in from 33 to 55 days, an average of 40 days, and adhesive plaster traction usually substituted. All traction was removed in from 53 to 104 days, and averaged 71 days. Four patients were using walking caliper braces before discharge from the hospital. One Steinman pin wound became infected but healed one week after removing the pin.

We know that solid union occurred in eight patients, nonunion in one, and one could not be followed after the sixtieth day, at which time he was walking with a caliper brace and had had firm union since the thirty-fifth day.

The case with nonunion is of particular interest in showing the possible value of the use of a walking caliper late in the treatment. The patient was

a Chinese, age 50, who was treated in suspension with a Steinman pin through the crest of the tibia. The weight was changed three times in the first 18 days in an attempt to improve the position. The alignment was good at the end of this time. The pin was removed on the fifty-fifth day and adhesive plaster traction substituted. Firm union is said to have been present on the sixtieth day. Traction was removed on the one hundred fourth day and he was allowed out of bed with a brace on the one hundred sixth day. He was discharged on the one hundred twenty-sixth day, walking well on a caliper with crutches. He stated later that he removed the splint as soon as he reached home. Shortly thereafter he felt the bone snap. However, he continued to bear weight without the caliper but with crutches. He failed to return for five months, at which time he had a definite false point of motion and the lower fragment of the oblique fracture was displaced proximally for about one inch and angulated outward. About ten months after injury a periarterial femoral sympathectomy was performed and the extremity was immobilized in a plaster spica with the fragments in fair alignment. There being no evidence of progressing union, 12 months after injury an open reduction with Lane plating was effected. The wound became infected and the plate was removed nine weeks after operation. A false point of motion was still present eight months later and the patient had a draining sinus.

The time of appearance of callus in the roentgenogram is mentioned on eight charts, varying between 21 and 100 days, averaging 42 days. The time of appearance of firm union is noted on seven charts, varying between 28 and 80 days, averaging 42 days.

We have the late results on six patients. Four were A_4, E_4, F_4, three of these being children. One was A_4, E_0, F_4, the opposite leg having been amputated previous to this injury. One was A_3, E_4, F_3, having some angular deformity and an indeterminate functional loss since he had a previous bony ankylosis of the knee joint. He was a peddler by occupation and was doing the same work at 13 months. Except for the three children, one A_4 result was treated by reduction and plaster encasement and one by ice tongs.

Closed reduction with the application of a plaster encasement was satisfactory in children. We believe immobilization in a plaster encasement usually offers little in adults. We found no method of traction generally satisfactory in adults and believe that open reduction should be used more frequently. If for some reason open operative reduction should not be performed, we favor skeletal traction by a Kirschner wire through the lower end of the femur, or through both the femur and the tubercle of the tibia, the latter to give traction and the former to overcome the posterior angulation. Adhesive plaster traction should not be used.

SUMMARY.—(1) One hundred-twenty consecutive patients with fracture of the shaft of the femur are considered.

(2) Seventeen early deaths are discussed, 14 of these being in multiple severe injuries. Three cases requiring amputation are discussed, one of whom died. These deaths and amputations are omitted from the rest of the study.

(3) One hundred one patients remained for more detailed analysis of treatment. Forty-eight of these were under 16 years of age. Forty-seven were the result of motor car accidents. There were but four compound and 20 comminuted fractures.

(4) Seventeen fractures were in the upper third, 74 in the middle third and ten in the lower third. The three groups are discussed separately.

(5) The primary hospital treatment was adhesive plaster traction in a Thomas splint suspension (43 cases), skeletal traction (35 cases), overhead adhesive plaster traction (11 cases), reduction and application of a plaster spica (11 cases), and open reduction (1 case).

(6) The initial treatment was unsatisfactory in 14 patients with adhesive plaster traction suspension and in two following reduction and application of a plaster spica.

(7) The final treatment, except for complications, was skeletal traction, 43; adhesive plaster traction in a Thomas splint suspension, 31; plaster spica, 12; overhead adhesive plaster traction, 11; and open reduction, 4.

(8) Traction methods were employed in 84 per cent of these patients.

(9) In the skeletal traction cases tongs were removed after an average time of 49 days.

(10) In adults, where traction only was used throughout the entire treatment, this was all removed after an average of 75 days.

(11) In the use of skeletal traction there were 21 infections in 42 patients. Fourteen of these healed promptly after removal of the tongs or pin. Seven, or 17 per cent, discharged for a long enough period so that we must consider that infection of bone was present.

(12) There were four early open reductions, three in simple fractures and one in a compound fracture. All were in children. One had no internal fixation, one kangaroo tendon, one wire, and one a steel plate. The compound fracture became infected, followed by osteomyelitis, but solid union occurred. The others remained clean. The incidence of open operation in children was eight per cent.

(13) Four patients sustained refractures, on the fifty-sixth, seventy-first and ninety-ninth days after injury, and one outside the hospital at nine months.

(14) Three patients had late open reductions for complications. One had an open reduction with steel plating on the seventy-sixth day for a refracture on the seventy-first day. Another had a bone transplant from the tibia on the seventy-eighth day for delayed union. The third had an internal fixation with a steel plate one year after accident for nonunion. The first two remained clean. The last became infected with persistent nonunion.

(15) There was one death during treatment. This occurred from embolism in a patient while in skeletal traction.

(16) Of the remaining 100 patients we know that 84 obtained solid union and one nonunion. The other 15 could not be followed, but there was no

reason, from the position of the fragments or the progress when last seen, to expect nonunion in any of these.

CONCLUSIONS

By these methods of traction, bony union with relatively little shortening and good functional results may be obtained in a high percentage of cases. Until our results were closely analyzed we had believed that they were good. They are not good enough. Overhead adhesive plaster traction was satisfactory in children under five years of age. Skin traction was unsatisfactory in children over five years of age, from the point of view of recovering original length. Skeletal traction in adults gave too high an incidence of infection. Therefore, in March, 1932, we changed to the use of Russell traction in all these groups. Sallick[1] has recently reviewed our experience for three years with this method. We are still not satisfied and believe that, in spite of the objections noted in the early part of the paper, we should perform more open reductions with internal fixation. This is particularly true in transverse fractures of the middle third and in most lower third fractures. Skeletal traction has been a marked improvement over reduction and application of a plaster spica or adhesive plaster traction with the Thomas splint. No single method of treatment will apply to all cases and one should be ready to employ each type of treatment according to the individual indication.

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Discussion.—Dr. Kirby Dwight (New York) said that one could not have listened to Doctor Kennedy's paper without realizing that the treatment of fracture of the femur has not yet become standardized or stereotyped. There has been a gradual, continual improvement, however, when one considers it in retrospect. Probably the history starts with what was called Buck's extension with a little railroad on the bed and with most of the traction applied to the bed. Then there was the period of the bone surgery when Lane plates were used, which can be subdivided into two periods, first the Lane plate with infection period; then the Lane plate without infection. The development of the Lane technic made a tremendous difference in that regard. Next came suspension and traction, with adhesive first, followed by skeletal traction by the three methods so commonly in use today: The Steinman pin, the calipers or ice tongs, and the Kirschner wire. Finally, there evolved Russell traction, on which so many looked skeptically at first only to embrace it for use in a good many types of fracture. During the 20 years' experience just reviewed, a good many other elaborate pieces of apparatus were developed for the treatment of this type of fracture, but in general, they were not adopted by surgeons. It was largely a question of expense in hospital work, presumably.

In the field of skeletal traction, the matter of the Steinman pin versus the Kirschner wire is current. The speaker personally regarded the latter as

far superior on account of the danger of infection with the pin. After the World War Doctor Dwight was stationed for some months in a hospital that cared for the soldiers returning with bone injuries. He learned a great deal about the healing of bone in the presence of infection, and one of the basic principles—in fact, one of the great truths in the treatment of old, infected compound fractures—is that a cavity or tunnel in bone that is larger than a certain minimum size cannot heal. It cannot because it cannot be first filled with healthy granulation tissue. The granulations forming on bone can reach only a certain thickness before their base becomes constricted by scar tissue, the blood supply to the surface becomes too little to combat infection, and there is unhealthy granulation tissue which continually discharges pus. Now the diameter of the Steinman pin, plus the thickness of the area of necrosis in infected cases, gives one a tunnel through the bone, the caliber of which is too large to be filled with healthy granulation tissue when the pin is removed. This is probably the main reason why the Kirschner wire does not behave that way: namely, because of its very much smaller diameter.

Regarding fractures in children between the ages of five and ten, the results are rather poor, poorer than in the years below five, or over ten. Bryant suspension is very satisfactory in young children up to the age of five. Above that age, there is a change in the child's physiology. He is no longer a baby, but a very active young animal whose activity puts on strength of muscle and takes off superfluous flesh so that the child of seven, for instance, has much greater strength of muscle in proportion to weight than has the four year old child. Therefore, in Bryant vertical traction, using both legs, the weight of the body is not sufficient to reduce the fracture. On the other hand, one cannot apply that traction to one leg because the child moves all around in bed, so that there is no immobilization of the fracture. At the same time, methods used in adults do not seem satisfactory in children of this age. They are pulled right down in the bed for the same reason: namely, that their body weight is light in proportion to the strength of their muscles.

One method used by Doctor Dwight for a good many years, the most satisfactory in his experience, is suspension by one leg at an angle of 45°, up to the frame above the foot of the bed, at the same time elevating the foot of the bed itself so that the body slides backward and traction is partly upward, so that one can get some of the effect of the Bryant traction, and partly longitudinal, so that the child is not twisted around from a traction applied to one leg. Doctor Dwight has used a sling as a kind of coaptive splint to suspend the thigh, and it need not extend below the knee. In general, he was in hearty agreement with the conclusions reached by Doctor Kennedy, with whom his experience largely coincided. In all cases that he can, Doctor Dwight uses Russell traction. In those where it is not successful he uses the other methods just stated. He has had some experience with fractures of the lower end of the shaft with angulation backward of the lower fragment. In one case treated with calipers he got the calipers a little high (calipers can act just as badly as the Steinman pin if one gets them in too far and a deep hole is made in the bone) and on account of the shape of the calipers the traction could not be purely longitudinal, but was upward and longitudinal. The immediate result was that it reversed the type of displacement, the proximal end of the distal fragment was brought anterior to the distal end of the proximal fragment. Doctor Dwight thought a long time as to what to do about that. Finally, experimenting, he saw that the caliper points were above the axis of rotation of the lower fragment. So he hung a weight on the pins of the caliper just as they emerged from the thigh, and was able to adjust the weight so that the lower fragment swung back into position.

Dr. Philip D. Wilson (New York) said that any study of results in a series of cases is of value chiefly when one makes a comparison with a similar group of cases. Such a study was available in the comparison of a series of 130 cases which had been analyzed from the Fracture Service of the Massachusetts General Hospital, with which he had been formerly associated; which cases had been treated in approximately the same manner.

Using 15 as the dividing age, there were about 50–50 adults and children. The main complications were: Slipping of the ice tongs, five cases; breaking of plates after operation, three; and postoperative sepsis, eight out of 26 open operations. Doctor Wilson thought that Doctor Kennedy had been well advised in his conclusion that because they were dealing with a large series of cases brought in off the streets—potentially infected—open reductions had been kept down to a minimum. In the series mentioned by Doctor Wilson there were 105 end-results obtainable in the 130 patients. There was shortening in 15.7 per cent of the children and in 30 per cent of the adults, but this shortening was not a measurement made at the time of the patient's discharge from the hospital, but a year—often two years—after discharge, when the patient returned for end-result observation. In some instances, it was made three years after discharge. To this difference in time of observation Doctor Wilson attributed differences in Doctor Kennedy's end-results and those found in his own series. Doctor Kennedy's chief difficulty was in children. Doctor Wilson thought he had not followed them long enough. The bones in children have a potentiality toward correcting deformity by growth. Cases of children discharged with no shortening may come back a year or two later with the leg an inch or so longer. This is not an argument to strive for shortening, but it is one against the endeavor to be too perfect. In general, children will give a good account of themselves.

Doctor Wilson defended the one leg overhead traction in small children, if the child is fixed on a Bradford frame so that he cannot wriggle around. It is desirable to get as much counter traction as possible. If the child is spring-boarded there is only half the counter traction one obtains when only one leg is slung up.

Dr. Clay Ray Murray (New York) emphasized two points made by Doctor Kennedy: One, that he was careful to define the type of hospital in which the results were analyzed; and secondly, he described the type of cases treated in that hospital. His conclusions, as drawn for such a situation, are accurate and could hardly be improved on. Doctor Murray also approved Doctor Kennedy's statement that the probability is that, in fractures of the shaft of the femur under modern conditions, the ideal form of treatment is open reduction, with early mobilization of the fracture. However, it is inadvisable to pursue such a course of treatment under ordinary conditions.

The truth of these two theories is rarely accepted, and it is regrettable that the method of treatment acknowledged as the right approach to the ideal under proper conditions is considered inaccessible in all except a very few select situations. Therefore, it is concluded that the less ideal methods should be employed in most instances. Doctor Murray thought that it was about time, with the development of surgical technic, that the difference between incidence of infection with the use of Kirschner wire over the Steinman pin is extended to the operative handling of fractures. If some of the time and individual effort that are put into finding means to avoid operating upon fractures, were to be put into finding means to make hospitals provide equipment and the organization to make such work safe, the disability after fractures of the long bones would materially decrease.

DR. FENWICK BEEKMAN (New York) said he had no idea that children comprised such a large number of cases in a series of fracture of the shaft of the femur. His personal experience had, however, been almost entirely with children, many of the cases having been admitted to the Children's Surgical Service at Bellevue Hospital. Here Russell traction had been employed for fracture of the femur in children for about the same period of time that Doctor Kennedy has used it at Beekman Street Hospital. Previous to 1932, when Doctor Beekman first commenced to use this method, children under six years of age were treated by overhead traction (Bryant traction) and older children were given an anesthetic, put on a Hawley table, the fracture reduced, and a circular plaster encasement applied from the hips down to the ankle, not including the foot. Skin traction having been previously attached, the traction was now applied by means of weights on the skin traction. The idea of this treatment was that the fracture was reduced by means of the Hawley table, and the plaster encasement and traction were used to keep the fragments in position. The results of a large number of children treated by this method have been reported by Dr. Carl Burdick. With the advent of Russell traction, this former method, in older children, was discontinued and the Bryant traction was applied only to children under three years of age.

Doctor Beekman favored Bryant traction applied to both legs, although he can remember some years back that it was frequently used on only one leg. Suspension, however, by both legs did not always keep the child quiet, as he had often seen a child, even at the end of a few weeks, with the two ropes twisted, having rotated the body three or four times. He did not believe that it was necessary to immobilize the pelvis in using Bryant traction.

He wished to stress the point which Doctor Kennedy had mentioned, that the traction does not, of necessity, reduce the fragments in fractured femora. In his experience he had found that hardly a child with Bryant traction had a perfect anatomic reduction, unless an anesthetic had been given and the fracture reduced before the suspension was applied. Doctor Beekman believed that this was also true of Russell traction. In most of the cases which had come under his observation, although much callus had developed, the fragments had not always been brought into exact end-to-end contact. Though union occurs with slight angulation, unexact alignment and some over-riding, the end-results after two or three years are excellent. At this time it is often impossible to tell by means of physical or roentgenologic examination which of the femora had been fractured. He had made the statement that, after reviewing a large series of cases, he felt that good anatomic and functional results could be obtained in almost all cases, even though there might be over-riding and lack of alignment of the fragments.

Doctor Beekman did not approve of open reduction in children. Out of some 200 cases of fracture of the shaft of the femur in children which he had seen during the last ten years, there had been but two open reductions performed in the Children's Surgical Service at Bellevue Hospital, and both of these were undertaken because of nonunion caused by the interposition of the muscles.

In discussing the length of time a child should be kept off his feet, Doctor Beekman said that he kept the patient in traction about an average of 42 days and then in bed, without splints, for about two weeks, after which the child was sent home with instructions to the parents that it be kept in bed. Though these instructions were always given, Doctor Beekman did not believe that they were always carried out, for in so many cases he found that as soon as the child left the hospital he was up and around. Even while still in the hospital, children who have just had traction removed have often been

seen to get out of bed and run about, but in none of these cases were there disastrous results. In the healing of fractures in children there is a great deal of callus thrown out. In Doctor Beekman's experience at Bellevue during 15 years, there were only about three instances in which refractures occurred in the femur.

The speaker thought it was timely that the subject of fractured femora should be brought before the profession, as the methods of treatment which have been recently introduced, such as Russell traction, have been far more satisfactory than those previously employed. He agreed with Doctor Murray that a surgeon should have recourse to more than one method of treating any particular type of fracture, as there were no two cases alike. Therefore, all cases cannot be treated in the same manner. A properly trained surgeon does not use standardized methods, but uses his judgment as to the best form of procedure to use in each particular case.

Dr. John A. McCreery (New York) thought it interesting to compare Doctor Kennedy's figures with those taken from the First Division at Bellevue Hospital, although these do not include children. There have been 150 fractures of the shaft of the femur treated on the First Division during the last 15 years, with a mortality of 14 per cent (21 deaths). This high mortality emphasizes the fact that fracture of the shaft of the femur is a serious injury with a high death rate due in part, perhaps, to the fact that in many of these cases there are complicating injuries. Seventeen deaths were early, the others occurring after varying periods of time, mostly as a result of infection. One hundred thirty-eight of the Bellevue fractures were simple; 18 compound, two of the latter being due to bullet wounds. In these the fractures were by impact rather than from penetration of the bone which was found in the high velocity war wounds. It was interesting to note how the type of treatment had gradually changed during the 15 years. At the start a good many cases were placed in a plaster encasement. Skeletal traction was then employed, first with the Steinman pin, then with ice tongs, and finally with a Kirschner wire. Doctor McCreery had had much the same trouble with infection in cases where tongs and pins were employed as did Doctor Kennedy in his series. Recently, Russell traction has been employed with increasing frequency, and with the same satisfactory results as noted by Doctor Kennedy. He was surprised to note how often open reduction had been resorted to in the series analyzed, his percentage fell between those reported by Doctor Kennedy and Doctor Murray. The usual method of immobilization has been with Lane plates. Seven of the 150 cases had come to amputation; most of these had been compound fractures involving the knee joint and in which infection had developed. Five cases had refractured; in all of these there had been some underlying pathologic condition such as bone cyst, Paget's disease or metastatic tumor.

WATER BALANCE IN NEUROSURGICAL PATIENTS

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In another article[1] attention has been called to the volume of blood lost during operations upon the brain, when a large bone flap is turned down. This is from two to five times the amount ordinarily shed in operations on the intra-abdominal and intrathoracic viscera. In addition to this excessive hemorrhage, which averages from 500 to 1,500 cc., the patient undergoing a prolonged operation for an intracranial tumor may lose up to 1,000 cc. of water from his skin and lungs. As a result of this loss of fluid from the vascular bed, sweat glands, and lungs, these patients often become seriously dehydrated.

The importance of transfusion during and after severe neurosurgical operations is generally appreciated, but in addition to this an understanding of water exchange is of practical value, because the replacement of fluid must be regulated with greater care after operations within the cranial cavity. The comatose patient is unable to drink, so that fluid intake must be regulated by the surgeon. Solutions administered intravenously or subcutaneously have a greater tendency to produce edema than liquids taken by mouth. If a slight excess of saline or glucose solution is infused after routine celiotomies, the resultant mild edema is not necessarily serious. But in operations upon the brain there is considerable tissue trauma, and a local increase in capillary permeability ensues. In addition to this, after a period of coma and malnutrition, the protein content of the plasma may already be reduced; with the further loss of protein contained in the liter or more of blood lost at operation, conditions for the production of cerebral edema are optimum. Swelling of the brain causes an obstruction to the outflow of blood from the venous sinuses and an increased production of cerebrospinal and edema fluid—a vicious cycle which may lead to death.

The purpose of this and another article[1] is to show the quantity of blood and other fluids commonly lost in the standard neurosurgical operations, what can be done to reduce this, and how the disturbed fluid balance can most effectively be restored.

Fluid Loss from Lungs, Skin, and Kidneys.—Rowntree,[2] and more recently Underhill[3] have reviewed the literature on water metabolism; accurate measurements of water balance in human beings have been made by Newburgh and his associates.[4] These studies have been applied to surgical patients by Coller and Maddock,[5, 6, 7] and there would be little justification for an investigation on the special aspects of water balance in neurosurgical pa-

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tients were it not for the fact that the total fluid loss in these cases is greater than any which has been reported heretofore. In order to recall the mechanism of postoperative water balance and to bring out its application to the special field of neurosurgery, we shall have to review very briefly the pertinent findings of the writers above mentioned and then record our determinations on this particular group of patients.

In healthy individuals, as well as in patients recovering from neurosurgical operations, there are three principal channels for the excretion of water: (1) Water in urine; (2) water in feces, and (3) water vaporized from skin and lungs.

Excessive fluid loss from diarrhea, vomiting, and draining wounds is fortunately such a rare complication after neurosurgical operations that it need not be considered here.

Water in the Urine.—Water excreted by the kidneys removes waste products from the blood, as well as certain substances such as sodium chloride and glucose present in excess of their normal threshold. In this connection it is important to point out that water is available for kidney secretion only after the skin, lungs, and intestinal tract have taken up their prior rights. A person on a low fluid intake in an excessively hot environment, or a patient with fever, vomiting, or diarrhea may become anuric when approximately 6 per cent of the body weight has been lost as water. During the recovery period after operation Coller and Maddock[6] recommend that the 24-hour volume of urine be maintained at a minimum of approximately 1,500 cc., with a specific gravity below 1.015. In dealing with patients after operation on the brain, where the possibility of cerebral edema is a constant source of danger, this would seem to be an unnecessarily generous estimate. It is our opinion that with normal kidneys a daily urine output of 800 to 1,000 cc. is sufficient, provided the specific gravity remains below 1.025. With a high fever or when renal function is impaired, a larger volume is required. Frequent determinations of the nonprotein nitrogen level in the blood are an ample safeguard against renal insufficiency. We have come to rely far more on the level of the nonprotein nitrogen than on the urinary volume or specific gravity. The latter may be distinctly misleading when intravenous fluids are administered. For example, when salt solution is given to a postoperative patient, much of the fluid may be retained in the interstitial spaces as edema. The tissues, therefore, may become overhydrated, while the kidneys are eliminating only a small volume of urine with a high specific gravity due to the increased concentration of sodium and chloride ions.

Water in the Feces.—Rowntree[2] has estimated that from 7.5 to 10 liters of fluid are poured into the gastro-intestinal canal daily by the salivary glands, the mucosal glands of the stomach and small intestine, and in the biliary and pancreatic secretions. In the normal individual nearly all of this is reabsorbed in the colon, so that in the absence of diarrhea, water lost in the feces rarely amounts to more than 200 cc. a day. The stool of normal consistency contains 50 to 80 per cent water.

*The Insensible Loss of Water.**—The third way in which water is eliminated from the body is by vaporization from the skin and lungs. Water lost in this way constitutes a most important part of the heat regulatory mechanism. In sick postoperative patients and in the presence of fever or extreme heat, the loss of water by this channel is frequently greater than in both the urine and feces. The extent of this loss, which the average surgeon leaves out of his calculations, is important because it continues even in advanced dehydration. Water continues to be vaporized from the skin and lungs even if none is left for kidney function. Water eliminated in this way accounts for the major part of the insensible loss of body weight, the other component being the weight of carbon dioxide exhaled in excess of the weight of oxygen absorbed. Since vaporized water may compose anywhere from 85 to 100 per cent of the total insensible loss of body weight, the error of assuming the insensible weight loss to be made up entirely of water is never great.

Newburgh, Wiley, and Lashmet[4] have found that normal individuals at comfortable environmental temperatures, and when performing only light work, vaporize an average of 1,000 to 1,500 Gm. of water daily. Under strenuous muscular exertion, during fever, or at temperatures over 80° F., the sweat glands become active and an increasing amount of water is vaporized to maintain a constant body temperature.

During the period of operation and the first few days thereafter, the amount of sweating is greatly increased. Fortunately in the course of craniotomies, especially when ether is not used, patients do not sweat as profusely as during the average celiotomy (Tables I and II). While the insensible loss, expressed as grams of water vaporized for each square meter of body surface per hour, is much less than in the routine celiotomy, the entire loss over the three to four hour period of operation may be approximately the same or somewhat more. Table I records the insensible weight loss in six patients during the course of intracranial operations. Patient 28, where ether was used, lost 1,012 Gm. This is 30 per cent greater than the highest recorded loss in Coller and Maddock's statistics, and occurred in spite of the fact that the lightest possible drapes were used. Patient 29, who underwent a more serious operation under avertin and local anesthesia, vaporized 574 Gm. In this patient, however, the huge additional loss of blood (2,050 cc.) resulted in the maximal total fluid loss of the series. The other four eliminated water vapor at a distinctly less rapid rate than major general surgical cases, although the longer operative period resulted in a total insensible weight loss almost as great as that observed in Coller and Maddock's cases (Table II).

* The detailed method of measuring the insensible loss of water is beyond the scope of this article. This has been described by Coller and Maddock.[5, 6] Briefly, the patient is transferred from bed to a delicate balance at the end of each period of observation. Over this interval all ingesta and excreta are accurately weighed. The total insensible loss of weight for each period is calculated by the following equation: Beginning Weight — (End Weight — Ingesta + Excreta). Since the Insensible Loss is measured by weighing the patient, it is better recorded in grams than in cubic centimeters.

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TABLE I

FLUID INTERCHANGE DURING PERIOD OF OPERATION*

Patient No.	Operation	Length of Operation—Hours	Period of Calculation—Hours	Anesthetic	Blood Loss	Vomitus	Urine	Insensible Loss and Sweating	Insensible Loss and Sweating—Gm./M²/Hr.	Total Fluid Loss
7.	Bone flap to evacuate subdural hematoma	2½	3¼	Local and ether	599	0	...	223	51	822
8.	Resection of meningioma of tuberculum sellae	2½	6	Local	573	0	...	241	28	814
13.	Partial section of fifth cranial nerve root	1	4	Local and evipal	86	0	...	127	22	213
28.	Partial removal of chromophobe adenoma of pituitary	2⅓	4⅔	Avertin-ether and novocain	1,200	0	113	1,012	109	2,325
29.	Subtotal resection of meningioma of wing of sphenoid invading orbit	2½	4½	Avertin and local	2,050	0	...	574	61	2,624
32.	Myoplastic craniotomy for oxycephaly	1¼	4	Avertin-ether and novocain	487	0	32	148	23	635

* All fluid measurements are recorded in grams, except blood loss, which is given in cubic centimeters. The operations were all performed in cool weather, with the operating-room temperature between 75° and 78° F. In patients Nos. 7, 8, 13, and 29 the urine was not obtained at end of operation, but was measured over the entire day.

TABLE II

FLUID LOSS DURING GENERAL SURGICAL OPERATIONS

Statistics of Coller and Maddock[5]

	Grams	Insensible Loss Gm./Sq. Meter/Hour
1. Partial gastric resection	533	127.5
2. Excision thyroglossal cyst	313	82.3
3. Exploratory, kidney	233	71.9
4. Repair inguinal hernia and appendicectomy	666	152.6*
5. Repair ventral hernia	328	88.3
6. Excision sarcoma of thigh	245	121.6*
7. Excision teratoid tumor of abdomen	180	62.2
8. Radical mastectomy	705	193.8
9. Partial gastric resection	560	149.8*
10. Colostomy	97	66.7
11. Repair inguinal hernia	259	167.6*
12. Repair ventral hernia	115	65
13. Hemorrhoidectomy	92	51.8*
14. Repair inguinal hernia	192	40.2
15. Appendicectomy	166	116.1*
16. Cholecystectomy	40	15 *
17. Appendicectomy	137	84.2*
18. Repair ventral hernia	347	113.9*

* Cases where ether was used.

These observations were made over a four-hour postoperative period.

The insensible weight loss during the early days after operation was measured in seven patients. Typical examples are given in Tables III and IV, which record the postoperative determinations after a myoplastic subtemporal decompression (Patient 32), and following a difficult removal of a large meningioma of the wing of the sphenoid (Patient 29). In Patient 32 the insensible loss after operation never rose above 1,561 Gm. per diem, the upper limit for a normal individual of 57 kilos. In Patient 29 over 2,700 Gm. of water were vaporized during the day after operation. As the temperature rose from 101° to 107.5° F., the loss from the skin and lungs diminished, because of the retention of heat which necessarily accompanies a rapidly rising fever.*

It is unfortunate for the surgeon in charge of the postoperative care of such a difficult case that there is no accurate index of early dehydration. Beyond the well known clinical signs, the most important laboratory data are: (1) Urine volume and specific gravity; (2) red blood count, hemoglobin, and hematocrit determinations; (3) serum protein; (4) nonprotein nitrogen; (5) body weight, and (6) blood volume.

We have depended on the first four tests, which are applicable to routine surgical patients, but wish to make it clear that none of these is infallible until

* This is only true over the period in which heat is being retained and the body temperature rising. When the fever is stationary or falling, the elimination of water from the skin and lungs is commonly increased.

TABLE III

PATIENT 32. MYOPLASTIC CRANIOTOMY FOR OXYCEPHALY

T. B., age 17, a mild oxycephalic, complained of headaches and diminishing visual acuity. Roentgenograms revealed complete closure of all his cranial sutures and deepened convulational markings indicative of a chronic increase in intracranial pressure. His visual acuity was diminished to 20/200 in each eye. There was moderate pallor of each optic disk, but no evidence of choking. His intelligence was nearly normal. The spinal fluid pressure was found to be slightly over 200 Mm. on two occasions. Decompression was recommended to reduce his headaches and to preserve vision.

2/23/37: A right myoplastic subtemporal decompression was performed by Doctor White, removing bone over a diameter of 6 cm. 1,244 Gm. of 5 per cent glucose in normal saline was administered during and after the operation, but there was no need for a transfusion. Blood loss at operation amounted to 487 Gm.

The patient made a satisfactory convalescence. There was only a slight febrile response. He was able to take fluid by mouth from the beginning and to eat solid food on the second day. Readjustment of his water balance after operation was therefore a relatively simple matter, calling only for the limitation of fluids to approximately two liters during the first three days. The reason for this patient's low serum protein (5.5 to 4.6 per cent—Table V) was never explained. With the resultant reduction in the colloid osmotic pressure there would have been a serious risk of cerebral edema if the patient had been comatose and required prolonged venoclysis (see protocol on Patient 30 below). There was no cause for concern here, however, as there had been no operative trauma to the brain and the patient was able to take a normal diet within three days.

	Weight of Patient without Dressing	Water Drunk	Intravenous	Food, Water of Oxidation, Medications	Urine	Specific Gravity, N. P. N.	Stool	Vomitus	Blood	Insensible Loss and Sweating	Maximum Mouth Temperature	Range of Room Temperature	Total Fluid Intake	Total Fluid Output	Fluid Balance
Preop. 2/12–2/13/37	57,570	904	0	1,277	727	1.026	196	0	0	1,368	101.0°	74 ↔ 72	2,181	2,291	110 −
Preop. 2/13–2/14	57,460 56,830	764	0	1,238	872	1.024 N.P.N. 24	204	0	0	1,556	100.2	74 ↔ 70	2,002	2,632	630 −
Day of Op. 2/23–2/24	56,870	794	1,244	323	294	1.032	0	447	487	1,173	100.8	73 ↔ 72	2,361	2,401	40 −
1st Day Postop. . 2/24–2/25	56,830	928	1,318	40	1,213	1.019 N.P.N. 17	0	0	32	1,561	100.4	68 ↔ 65	2,286	2,806	520 −
2nd Day Postop. 2/25–2/26	56,310	1,554	0	479	2,326	1.015	0	0	0	1,263	99.9	72 ↔ 66	2,033	3,589	1,560 −
3rd Day Postop. 2/26–2/27	54,750	1,189	0	954	1,891	1.008	0	0	23	939	99.8	76 ↔ 68	2,143	2,853	710 −
4th Day Postop. 2/27–2/28	54,040	1,189	0	1,273	1,176	1.016	0	0	0	1,266	99.0	70 ↔ 66	2,462	2,442	20 +
5th Day Postop. 2/28–3/1	54,060	1,189	0	1,275	1,295	1.019	154	0	0	1,305	99.8	74 ↔ 68	2,464	2,754	290 −
6th Day Postop. 3/1–3/2	53,770 54,070	1,188	0	2,827	1,196	1.021	0	0	0	1,519	99.9	76 ↔ 70	3,015	2,715	300 +

Weighings all performed at 7:00 A.M.

The patient's weight and all fluid measurements except blood are recorded in grams. Blood lost at operation is given in cubic centimeters.

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dehydration has reached the advanced stage in which blood volume begins to fall. From these criteria, as well as from her clinical appearance, Patient 29 did not appear to be dehydrated (Tables IV and V). Judging from her rapid loss of weight, however, and by her large daily excess of fluid output over intake, we feel that she must have become so. Although it would have been impossible to detect this by any tests which can be made outside of a special metabolism ward, we believe that the rules for postoperative fluid replacement which are detailed below are adequate to prevent this dangerous complication without risk of cerebral edema.

In concluding this subject of water loss after operation, the importance of the insensible loss cannot be emphasized too strongly, because of its large volume and the fact that, unless it is recognized and adequately replaced, it will continue until no water is left for renal secretion. Contrary to what is still assumed by many surgeons, it is impossible to obtain a complete picture of the total water exchange by a comparison of the fluid intake with the urine output. An understanding of the way the body eliminates fluid, and the volumes concerned, is essential for the maintenance of an adequate intake in the sick or comatose patient.

Methods of Reducing Water Loss.—In addition to controlling hemorrhage in the patient on the operating table, it is important to reduce the insensible loss of water from the skin and lungs. For this purpose, as well as for the reduction of hemorrhage, it is our belief that ether anesthesia should be avoided whenever possible. This statement is borne out by a comparison of the insensible loss per square meter of body surface per hour in etherized and nonetherized patients (Table I), and by observation of the relatively slight degree of perspiration which is commonly present after prolonged craniotomies under local anesthesia.

Besides selecting an anesthetic which will not increase the activity of the sweat glands, it is essential in warm weather not to overburden the patient with thick coverings. In preparation for operation it is routine practice at many hospitals to cover patients with a blanket and warm leggings, before numerous additional layers of sterile drapes are superimposed on the operating table. This practice is pernicious if the temperature in the operating room is over 80° F.; Coller and Maddock[5] have shown that the insensible loss of water during operations in hot weather can be considerably reduced when the lightest possible drapes are used. In all of our cases in Table I, where the temperature in the operating room averaged between 75° and 78° F., the lightest possible gown, leggings, and drapes were used. The same procedures were applied after operation. In hot weather a special effort should be made to keep the sick postoperative patient from losing still more of his depleted water supply by sweating.

Methods of Restoring Water Loss.—In the normal individual the fluid intake and output are so regulated that the water content of the body is maintained at approximately a constant level. This relationship may be upset in a surgical patient who is too sick to have a normal sensation of thirst and is

unable to take an adequate amount of fluid by mouth. In such a patient the water intake must be regulated by the surgeon, and recovery may depend on how skillfully this is done. The fundamental aspects of fluid intake and the replacement of water in dehydration have been discussed by Coller and Maddock,[7] so that only a short review of this subject, with particular emphasis on the special problems which come up in neurosurgical patients will be presented here.

In brief, there are two main sources of water intake: (1) Water drunk, and (2) food, either eaten in the diet or body tissue consumed.

The average adult drinks from 800 to 2,000 cc. a day. In the water which comes from metabolism there is no distinction between food eaten and body tissue burned. The water content of vegetables and fruits averages 80 per cent of their weight, while meats contain from 60 to 70 per cent water. There is an additional source of water derived by the process of metabolism known as the water of oxidation. Two hundred to 400 cc. of water are furnished daily from this source. In a mixed diet, each gram of food eaten yields through its water content plus its water of oxidation around 0.9 Gm. of water. Calculation of the water derived from the routine house diet at the Massachusetts General Hospital eaten by the average patient in a single day shows:

Total weight of food		1,885
Water content of food		1,400
Water of oxidation	Protein	29
	Fat	93
	Carbohydrate	133 +
Total		1,655 Gm.

In the house diet listed above are included:

Milk	500
Cream	90
Soup	150
Coffee and tea	0 Gm.

When the patient is unable to take anything by mouth, the only water available to compensate for the large daily deficit is that coming from the body glycogen, fat, and protein burned for energy. This yields about 500 cc. daily. The interstitial fluid amounts to about 15 per cent of the body weight and constitutes a reserve which can be drawn upon quite extensively in defense of the blood plasma volume without disturbing the organism. Moderate reduction of interstitial fluid (dehydration) is harmless and probably desirable, but when up to 6 per cent of the body weight is lost, serious effects begin to develop.

The application of these statistics to the restoration of fluid balance after intracranial operations is brought out by the data of typical cases in Table I. During the operative period the average patient will lose from 500 to 1,500 cc. of blood, and from 200 to 1,000 Gm. of water are vaporized from the skin and lungs. The secretion of urine during this period is small, averaging less

than 100 Gm. during an ordinary craniotomy. The total loss of fluid, therefore, is usually more than 1,000 Gm. and may even exceed 2.5 kilos. Hemorrhage and dehydration are frequently severe enough to require immediate attention, the replacement by infusion or by transfusion, depending on the blood loss and the degree of shock. Our customary procedure has been to start an intravenous infusion of 5 per cent glucose in normal saline at the beginning of all extensive operations and to permit 500 to 1,000 cc. to run in slowly during the course of the operation. As has been described in the preceding article,[1] citrated blood may be substituted for the infusion at any time if there is an alarming fall in the blood pressure. When it is estimated that a hemorrhage amounting to a liter or more has taken place, a transfusion is given in the operating room. When larger amounts of blood have been lost and the blood pressure remains low, another transfusion is given in the ward. By following this procedure the fluid deficit after operation should rarely exceed 1,000 cc. The intravenous infusion is continued as the patient is returned to the ward and a total of up to 2,000 allowed to run in slowly during the course of the next four hours. This is all that should ordinarily be given during the day of operation, as a larger volume subjects the patient to the risk of cerebral edema.

Conditions in postoperative neurosurgical patients being no different from those in ordinary surgical cases, we have found that the data in our patients during the recovery period agree with those reported by Coller and Maddock.[6] In neurosurgical cases, however, the matter of continued fluid replacement requires far greater care than in the ordinary surgical patient, because any excessive ingestion of fluid adds to the ever present risk of cerebral edema. The dangers of cerebral edema after brain trauma have been emphasized by Fay,[8] who has also pointed out the importance of a limited fluid intake under these circumstances.

Patients with tumor of the brain frequently have been through a period of malnutrition before entering the hospital, so that their serum protein is depleted. With a hemorrhage of 1,500 cc. at operation they may suffer a further loss of serum protein of 50 Gm. Stewart and Rourke,[9] who have studied the effects of comparable acute hemorrhage in dogs, have observed a decrease in serum protein ranging from 0.5 to 1 per cent. The consequent reduction in the colloid osmotic pressure of the blood, in conjunction with the presence of an area of traumatized brain tissue where the capillary walls are injured and abnormally permeable, predisposes to cerebral edema. If the blood is further diluted by the infusion of excessive amounts of saline solution, edema is especially likely to follow. This tendency to edema formation after a reduced protein intake, operation, and infusion has been pointed out by Jones, Eaton, and White[10] and more recently by Mecray, Barden, and Ravdin.[11] While the so-called "edema level," with the plasma protein reduced to 5.2 per cent, is not often reached, it should be borne in mind that any reduction of the serum protein means an increased tendency towards the retention of fluid in the interstitial spaces, especially if sodium salts are in-

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TABLE IV

PATIENT 29. SUBTOTAL RESECTION OF MENINGIOMA OF WING OF SPHENOID

A. Z., age 52.—In 1931, the patient noticed swelling in her left temple just behind the eye and also slight protrusion of the eyeball. She first entered this hospital in 1933 without any subjective symptoms or neurologic signs. Roentgenograms showed the characteristic changes of a meningioma arising from the sphenoid bone. On 8/29/33 a subtotal removal was performed by Dr. W. J. Mixter, from which the patient made a rapid convalescence.

It had been impossible to remove the extension of the tumor into the orbit, and as a result the patient noticed increasing exophthalmos. She remained free from symptoms, but reentered the hospital in June, 1936, with the onset of chemosis of the conjunctiva.

A second subtotal removal of the meningioma was carried out by Doctor Mixter 6/23/36. His operative notes are given verbatim:

"The old scalp flap was turned down. There was very free bleeding. The dura was freed up and the wing of the sphenoid was exposed, and found to be infiltrated with new growth. The dura was opened and the growth was found to be straddling the wing of the sphenoid, compressing the temporal and frontal lobes. On account of the extensive vascularity of the lesion it was not deemed advisable to remove it in toto. Most of the growth back of the sphenoid was scooped out. There was very free bleeding from this region, which was partially controlled with muscle packs, and the scalp flap was replaced without drainage."

Immediately after the operation there was considerable staining on the dressing, but the patient remained in fair condition and free from shock. On the day following operation she lapsed into a semicomatose condition and developed Cheyne-Stokes type of respiration. Lumber puncture at this time showed a pressure of 135 Mm. and a bloody spinal fluid; on the day before she died the spinal fluid pressure had risen to 300 Mm. On the second and third postoperative days she developed deepening coma and a rising temperature, which reached 107.5° F. at death on the morning of the fourth day after operation. No autopsy was permitted.

	Weight of Patient without Dressing	Water Drunk	Intravenous	Food Water, Including Water of Oxidation	Urine	Specific Gravity	N.P.N.	Stool	Vomitus	Spinal Fluid	Blood	Insensible Loss and Sweating	Maximum Mouth Temperature	Maximum Room Temperature	Total Fluid Intake	Total Fluid Output	Fluid Balance
Day before Operation 8:00 A.M. 6/21/36 to 8:00 A.M. 6/22/36	109,970	1,079	0	1,693	722	1.015	29	0	0	0	0	2,275	98.8°	84°	2,773	2,998	225 −
Day of Operation 8:20 A.M. 6/23/36 to 7:15 A.M. 6/24/36	109,250	265	1,036*	0.9	664	1.019	25	0	0	0	2,050	1,233	101	70	1,302	3,948	2,646 −
1st Day Postop. 7:15 A.M 6/24/36 to 7:15 A.M. 6/25/36	106,604	78	919	51	585	1.019	30	0	81	12	72	2,748	101	70	1,048	3,500	2,452 −
2nd Day Postop. 7:15 A.M. 6/25/36 to 7:15 A.M. 6/26/36	104,101	0	1,044	28	781	1.024	26	0	0	10	22	2,340	103.5	81	1,072†	3,153	2,080 −
3rd Day Postop. 7:15 A.M. 6/26/36 to 7:15 A.M. 6/27/36	101,991	0	3,300	91	756		30	0	0	0	23	1,892	107.5	85	3,391	2,671	720 +
4th Day Postop. 7:15 A.M. 6/27/36	102,619	Died at 8:57 A.M.															

The patient's weight and all fluid measurements except blood are recorded in grams. Blood lost at operation is given in cubic centimeters.

* Includes transfusion of 575 cc.

† Intravenous of 3,000 had been ordered, but through an oversight the full amount was not given.

fused. If the patient has been undernourished and has a serum protein content of under 5.6 per cent, the deficiency should be corrected before operation. This can be done easily in the patient who is able to eat by increasing the protein content of the diet. Recent work by Field,[12] which is not yet published, has shown that vitamin B, in the form of Lederle's liver extract intramuscularly and Harris's yeast extract by mouth, is of considerable help in raising a low serum protein level. In the sick or comatose patient blood transfusions are the only available method of increasing the protein content of the plasma. After operation, a patient who remains comatose for a long period must be watched for any significant reduction in the plasma protein. When this is discovered, treatment by transfusion or duodenal feeding should be started. Unfortunately these patients, even if comatose, are usually too restless and uncontrollable to tolerate nasal feedings. Patient 30 illustrates this situation.

Case Report.—(Patient No. 30) : F. M., male, age 53. A disoriented patient with three months' progression of headaches, nausea, vomiting, loss of vision and memory. Examination showed weakness of right face, a marked aphasia predominantly of the receptive type, slight papilledema, and a right homonymous hemianopsia. There were no motor weakness or changes in reflexes, but position sense and stereognosis were impaired on his right side. Lumbar puncture gave an initial pressure of 250 and a protein of 750 mg.

A preoperative diagnosis of left occipito-temporo-parietal glioma was made, and operation performed February 4, 1937, by Doctor White. Exploration revealed a deeply situated glioblastoma multiforme in the area predicted. A large amount of this was sucked away to form an "internal" decompression, and the bone flap replaced. The patient was given a transfusion of 550 cc. of citrated blood, after an infusion of 600 cc. of 5 per cent glucose in normal saline, and returned to the ward in good condition. The blood loss at operation amounted to 753 cc.

On the day after operation the patient was in excellent condition and took 1,800 cc. of fluid by mouth. At this time the report of his preoperative blood chemical analyses was received (Table V). This showed a serum protein of only 5.1 per cent, which was unexpected as he had denied any dietary deficiency. His wife subsequently stated that he had eaten no meat for a month and that he had had a very poor appetite during this period. During the following days his condition became progressively worse, with uncontrollable restlessness followed by deepening coma. His daily fluid intake averaged 1,700 cc. of 5 per cent glucose in normal saline by venoclysis. Feeding by nasal catheter was impossible because of his lack of cooperation. Slight edema of his face and neck were noted on the fourth day, and his serum protein had fallen to 4.7 per cent, after a rise to 5.3 per cent following the transfusion at operation. He was therefore transfused again. But the swelling of his face and neck continued to increase and on the seventh postoperative day slight edema appeared over his sacrum and ankles. At this time his serum protein had risen to 5.4 per cent, his serum sodium was normal, but the chloride elevated to 109 milliequivalents (the first time it had been above normal). The patient was in profound coma with absent pupillary responses and beginning Cheyne-Stokes respiration. His spinal fluid pressure had risen to 700 Mm.

A diagnosis of cerebral edema was made and the patient reexplored, a week after the first operation. On turning down the bone flap the swollen brain bulged under great pressure. The bone flap was therefore sacrificed and the scalp resutured. A third transfusion was given and a nasal catheter inserted into his stomach. In this way it was possible to give increasing feedings rich in protein. No further saline was administered. On this regimen and after a fourth transfusion, the serum protein rose to 5.7 per cent,

consciousness was rapidly regained, and within five days the patient was able to take a solid diet. Coincident with this clinical improvement his subcutaneous edema disappeared.

The only other patients who were found to have significantly low serum proteins before operation (5.5 and 5.2 per cent) developed further postoperative reductions to 4.6 and 4.9 per cent (Patients Nos. 32 and 35, Table V). Neither gave histories of dietary deficiencies. Patient No. 32 happens to be one of the seven subjects upon whom complete postoperative fluid balance studies were carried out (Table IV). These statistics give no indication of water retention, as there was a good urinary secretion and a negative fluid balance throughout the period of observation. Patient No. 35, after a radical resection of a large frontal meningioma, also showed no signs of edema. Fortunately in both instances the patients were able to resume a normal diet within a few days. Had they become comatose and required intravenous infusions, both might have developed this complication.

For the reasons enumerated above the problem of restoring water balance in the neurosurgical patient, who is unable to take solid food or even water by mouth, becomes a difficult one. The surgeon must estimate the fluid needs of his patient with care, and supply the caloric needs as well as he can. Subcutaneous infusions cause unnecessary discomfort, and absorption by proctoclysis is too uncertain. Venoclysis, therefore, is frequently the only available method of administering fluids and nourishment to sick postoperative patients.

The dependence of water balance on normal quantities of sodium and chloride ions in the blood and tissue fluids has been described by Gamble.[13] In contrast to the recovery period after operations on the gastro-intestinal tract, neurosurgical patients rarely vomit more than minimal amounts. Yet the data recorded in Table V demonstrate that a moderate reduction in concentration of serum sodium often occurs. These patients suffered a loss of base up to ten milliequivalents, while the concentration of chloride ion varied from a slight rise to a fall of nine milliequivalents. Such reductions in sodium to 126 and in chloride to 97 milliequivalents correspond to figures obtained in patients after thoracoplasties by Stewart, Talbott, and Churchill.[14]

In the absence of vomiting, diarrhea, or wound secretions, these sudden postoperative fluctuations in the concentration of sodium and chloride ions in the blood serum can be accounted for in part by loss through the sweat glands. These patients at rest in bed and at room temperatures ranging from 70° to 75° F. rarely showed visible sweating, yet it is known that an extensive loss of salt can take place through the skin in other febrile conditions. A further explanation for shifts in the concentration of sodium and chloride ions in the blood has been suggested by a recent investigation of Stewart and Rourke[9] on the effects of hemorrhage on the electrolyte balance. In their experiments dogs were bled an amount corresponding to the severer grades of hemorrhage encountered in neurosurgical operations. As in our postoperative statistics, which are recorded in Table V, they observed that the serum sodium fell distinctly, while variable changes took place in the

TABLE V

CHANGES IN THE BLOOD CHEMISTRY AFTER CRANIOTOMY

	Patient 7. Parietal bone flap and evacuation of subdural hematoma. Blood loss: 599 cc.			*Patient 13.* Partial section of fifth cranial root. Blood loss: 86 cc.			*Patient 29.* Subtotal resection of meningioma of sphenoid. Blood loss: 2,050 cc.			*Patient 30.* Partial resection of glioblastoma multiforme. Blood loss: 753 cc.				*Patient 31.* Negative cerebellar exploration. Blood loss: 927 cc.				*Patient 32.* Myoplastic subtemporal decompression for oxycephaly. Blood loss: 487 cc.			
	Protein	Sodium	Chloride	Protein	Sodium	Chloride	N. P. N.	Protein	Chloride	N. P. N.	Protein	Sodium	Chloride	N. P. N.	Protein	Sodium	Chloride	N. P. N.	Protein	Sodium	Chloride
Preop.	5.7	136	105	5.9	...	102	29	5.8	102	29	5.1	138	101	20	6.4	137	106	24	5.5	139	105
Postop.	6.9	...	103	5.8	131	101	Transfusion			Transfusion				Transfusion							
1st Day							25	6.1	105												
2nd Day							30	5.9	101	26	5.3	138	101			132		17	4.6	138	109
3rd Day	6.6	131	97	5.5	136	100	26	6.2	109												
4th Day							30	5.7	...						5.7	132	101	..	5.5	...	103
							Died on 4th day.			18	4.7	130	105								
6th Day	5.7	...	100							Transfusion											
7th Day										17	5.4	139	109	17	5.8	138	104	19	5.2	135	106
8th Day										Transfusion											
9th Day	6.1	142	103							28	5.3	136	104	Transfusion							
10th Day										Transfusion											
12th Day	6.0	142	106							29	5.7	128	99								
Comments:	Good convalescence.			Good convalescence.			No solid food postop. Coma on 3rd day. Total venoclyses of 6,299 Gm. 5% glucose in normal saline. See Table IV.			Onset of coma on 3rd day. 6,800 Gm. 5% glucose infused in next four days. Edematous and moribund on 7th day. Recovery after decompression of edematous brain and high protein intake.				Good convalescence.				Good convalescence. Resumed solid diet on 2nd day and developed no edema. See Table III.			

TABLE V (*Continued*)

	Patient 33. Parietal bone flap and evacuation of subdural hematoma. Blood loss: 918 cc.				*Patient 34.* Negative cerebellar exploration. Blood loss: 708 cc.				*Patient 35.* Total resection of frontal meningioma. Blood loss: 1,187 cc.				*Patient 36.* Frontoparietal exploration and biopsy. Blood loss: 1,050 cc.				*Patient 37.* Subtotal resection of frontoparietal astrocytoma. Blood loss: 1,289 cc.			
	N. P. N.	Protein	Sodium	Chloride	N. P. N.	Protein	Sodium	Chloride	N. P. N.	Protein	Sodium	Chloride	N. P. N.	Protein	Sodium	Chloride	N. P. N.	Protein	Sodium	Chloride
Preop.	26	7.5	137	101		5.9	137	99	19	5.2	138	109	21	5.8	135	98	24	5.6	136	107
Postop.					Transfusion				Transfusion				Transfusion							
1st Day	24	6.3	131	99	35	5.9	143	103	18	5.5	140	107	37	5.7	138	101	23	5.3	136	101
2nd Day																				
3rd Day					32	5.9	138	100	19	4.9	136	109	33	5.3	140	100	25	5.8	137	98
4th Day	19	5.3	133	98																
6th Day									..	5.0	139	105	36	6.3	126	98	29	5.3	136	111
7th Day	18	6.2	137	99	32	5.6	136	101												
8th Day	Transfusion																			
9th Day																				
10th Day																				
12th Day															133	99				
Comments:	Good convalescence.				Good convalescence.				Good convalescence. Able to resume normal diet on 2nd day and showed no tendency to edema formation.				Developed transitory hemiplegia, but made complete recovery. The drop in Na. and Cl. between 3rd and 6th days is due to $MgSO_4$ dehydration and diarrhea.				Transitory hemiparesis, but recovered fully.			

All determinations were made on blood serum.
Analyses are recorded in the following terms:
Serum nonprotein nitrogen: milligrams per cent, by method of Folin (Laboratory Manual of Biol. Chem., D. Appleton & Co., 1927).
Serum protein: per cent, by modification of method of Folin and Denis (Jour. Biol. Chem., **26**, 473, 1916).
Serum sodium: milliequivalents, by method of Butler and Tuthill (Jour. Biol. Chem., **93**, 171, 1931).
Serum chloride: milliequivalents, by Van Slyke method, modified by Fiske (personal communication).

chloride level. With this they found a sharp increase in urinary potassium. Potassium being the principal intracellular base, its increase in the urine must signify that in compensating for blood loss fluid is drawn into the circulation from tissue cells as well as from interstitial areas. Since interstitial fluid contains a lower concentration of sodium than plasma and the fluid within the cells contains none, the inflow of fluid from these reservoirs must necessarily reduce plasma sodium. On the other hand, the serum chloride level may rise or fall, depending on whether water is withdrawn chiefly from the interstitial spaces, where its concentration is higher than in the plasma, or from the cells themselves, which contain none.

Although these chemical changes are not of sufficient magnitude to require replacement in order that the tissues may retain a safe level of hydration, it is important to point out that our lowest values correspond to those found in early Addison's disease. Welch, Masson, and Wakefield[15] have reported that a reduction in the blood electrolytes of this degree from an intestinal fistula may be accompanied by symptoms of weakness, drowsiness, and a low blood pressure, but no such symptoms were observed in cases of heat cramps reported by Talbott,[16] where even lower levels were found. It has been quite impossible to correlate the postoperative condition of our patients with the level of sodium and chloride ions in the blood serum. Patient No. 36, for instance, failed to show any symptoms referable to salt depletion when his serum sodium and chloride had fallen to 126 and 98 milliequivalents respectively after a five-day period of magnesium sulphate dehydration and diarrhea (Table V). There is no question that the severe disturbances in blood electrolytes which occur in vomiting and diarrhea must be restored by saline infusions, and it must be remembered that there may be a significant loss of salt from the bowel in magnesium sulphate dehydration. But from our present lack of evidence that slight reductions in sodium chloride are harmful per se, any effort to correct symptomless minor deficiencies seems unnecessary.

The risk of edema formation inherent in the administration of saline solutions has been pointed out by Coller, Dick, and Maddock.[17] These investigators have shown that a reduction in renal secretion and retention of water in the tissues will result when there is an excess of sodium. Furthermore, they report that in a group of sick surgical patients given 5 per cent glucose in a physiologic solution of sodium chloride, all retained water and gained weight. When the intravenous solution was changed to 5 per cent glucose in distilled water, diuresis took place and the retained water was promptly given up. In Patient No. 30 (see above), where glucose in normal saline was given by venoclysis over a period of six days, generalized edema developed in the presence of a low serum protein and an elevated chloride (109 milliequivalents) and the patient nearly died of edema of the brain. Prior to this dramatic experience and the excellent article by Coller, Dick, and Maddock, we had not been sufficiently aware of the risks inherent in the use of normal salt solution.

Edema from too much salt solution is obviously a very real danger. For this reason we recommend limiting the routine use of glucose in saline to the

period of operation, during which there is a considerable loss of sodium chloride. Thereafter, fluids can usually be taken by mouth in adequate volume by neurosurgical patients. Saline solutions should only be used when the serum electrolytes are seriously depleted by prolonged vomiting or diarrhea, and then in limited amounts. For a comatose patient 5 per cent glucose in distilled water is the best solution for intravenous use. Glucose so administered is rapidly oxidized and yields 3.75 calories for each gram burned, as well as 0.6 Gm. of water of oxidation. It prevents starvation ketosis and, in addition, the water left is available for renal secretion.

Having described the essential features which govern water exchange in neurosurgical patients, we must now formulate specific rules for the prevention of dehydration and the restoration of an upset water balance once it has taken place. The first requisite is to make sure of the function of the patient's kidneys and his state of hydration before operation. If there has been a period of malnutrition, a low plasma protein must be raised by a diet high in protein and in vitamin B^{12}; or by blood transfusion if operation must be performed at once. Similarly, after a period of vomiting or diarrhea the altered balance in blood electrolytes must be corrected. Regardless of whether there is an excessive depletion of sodium or chloride, the normal equilibrium can be restored by infusions of isotonic salt solution, provided renal function is adequate to handle an excess of either ion. Before operation there is usually no need of limiting fluids by mouth, and it is a good working rule to send no patient to the operating room unless he has passed over a liter of urine on the day preceding the operation.

After operation, every patient who has been put through a major intracranial operation is dehydrated. Our method of giving an intravenous infusion of 5 per cent glucose in normal saline during every extensive operation and one or even more transfusions, whenever an excessive amount of blood has been lost, has been described above. When the patient has been returned to the ward, a chart should be kept of the fluid intake and the output of urine and vomitus, as well as an approximate estimate of the amount of sweating. It has already been pointed out that a comparison of fluid intake with urine output fails to give a complete picture of the water balance, because it neglects the insensible loss of water vaporized from the skin and lungs. In the sick postoperative patient this is usually the largest factor in the fluid loss. In calculating the fluid needs of a patient, it is necessary to make an approximate estimate of the insensible water loss and then to add it to the volume of the urine, vomitus, and feces. It is safe to assume a minimum insensible loss of a liter to 1,500 cc. each 24 hours in a patient with a normal basal metabolic rate who is afebrile and in a room with a temperature below 80° F. (Table III). In very hot weather, or with a fever of over 104° F., the insensible loss may rise to a maximum of over 2.5 liters (Table IV). To maintain a minimum safe urinary secretion (700 to 1,000 cc.) in an adult patient with normal kidneys, the fluid intake must therefore exceed 1.5 liters. Two liters is perhaps a safer minimum daily allowance for patients who do not require a limited intake. Any further reduction in water intake can only be made

with safety if a daily watch is kept to make sure that the specific gravity of the urine does not reach the kidneys' upper limit of concentration* and that there is no hemoconcentration or retention of nitrogen in the blood. In the presence of diseased kidneys which are no longer able to excrete a concentrated urine, and also in febrile patients where increased waste materials must be eliminated, a greater fluid intake will be necessary in order to maintain an adequate secretion of urine.

In postoperative neurosurgical patients who are able to take adequate fluid by mouth, water balance is easily maintained. During the early days of convalescence it is advisable simply to limit fluids to the lowest safe level (1,500 to 2,000 cc. daily from all sources in the afebrile patient with normal kidneys) in order to reduce intracranial pressure to a minimum. To accomplish this it is necessary to make a rough estimate of the water content of the food eaten (refer to the figures at beginning of this section), as well as to chart the liquids drunk, balancing intake from both sources against the large insensible loss of water from the skin and lungs and the necessary excess to supply the fluid needs of the kidneys. The figures from such a case are given in Table III.

The data in Table IV serve to emphasize the large amounts of blood and fluid which can be lost in the course of an extensive operation for brain tumor, as well as the large volumes of water which can be eliminated during the postoperative course in the presence of a high fever. Here the limited ability of the patient to take fluid by mouth had to be supplemented by the intravenous route. To have maintained a conservative level of hydration she should have received an intake of over 2,500 cc. daily, but through an error a smaller amount was given. This protocol is therefore not a fair example of adequate fluid replacement. In spite of the low intake, the patient gave no clear-cut evidence of dehydration, aside from a 6.7 per cent reduction in body weight. We wish to stress the point that depletion of the interstitial fluid reservoir can proceed to a considerable extent before it can be detected by the clinical appearance or by laboratory tests. Characteristic changes cannot be relied on until the blood plasma volume begins to fall and a serious state of dehydration makes its appearance. One can therefore safeguard water balance more effectively by appreciating the fluid needs of the patient than by the most careful watch for signs of dehydration. Complete postoperative measurements of fluid balance which have been made in five additional cases have shown no essential differences from those recorded above.

We realize that we are reducing the margin of safety in recommending such a limited replacement of fluid, which is somewhat more generous than that advocated by Fay[8] for the treatment of traumatic brain injuries, but distinctly lower than that recommended by Coller and Maddock[6] for patients after ordinary surgical operations. Granted, however, that the surgeon recognizes this risk and is willing to follow his patient by the quantitative tests

* In the patient with normal kidneys able to concentrate urine to a specific gravity of 1.030, we have felt that readings below 1.025 usually signify an adequate state of hydration.

outlined above, we believe that a limited degree of hydration is much safer after operative trauma to the brain than any excess of fluid. Blood studies on patients treated in this way have shown no evidence of nitrogen retention, no rise in the erythrocyte count, nor any increase in the hemoglobin, hematocrit, or serum protein indicative of blood concentration.

Discussion.—The physiology of water balance is of extreme importance to the neurosurgeon because he is faced by the problem of severe dehydration after so many of his operations. In addition to the large amount of blood lost in the course of an extensive operation for brain tumor, the loss of water from the skin and lungs is at times greater than during the average abdominal operation, although the loss per hour is distinctly less.

For these reasons the patient is nearly always dehydrated during the early recovery period. We have, in fact, observed a total fluid loss of 2.5 liters in the operating room. In a delirious febrile patient a corresponding amount may be lost daily. Without effective replacement such a loss can be tolerated for only a limited period without producing symptoms of severe dehydration. Coller and Maddock[7] have shown that this train of events develops when over 6 per cent of the body weight has been lost as water. On this basis a patient who weighs 100 pounds (45.5 Kg.) cannot afford to lose much over 2.5 liters, nor a 150 pound (68.2 Kg.) individual over four liters of body fluid reserve without developing serious complications. Munro[18] has observed semicomatose patients after severe intracranial injuries who have been treated by intensive dehydration. After a prolonged period on an inadequate water intake such cases may develop increasing mental confusion, a rising fever, and a progressive fall in urine volume and spinal fluid pressure to below 50 Mm. of water. He believes that this syndrome represents a toxic state of dehydration and has given such individuals over 5,000 cc. of fluid daily. Coincident with the rise in urine output he has observed a striking clinical improvement.

While the unfavorable sequelae of dehydration are obvious, the deleterious effects of overhydration are less commonly recognized. The general surgeon can safely ensure an adequate intake by allowing a moderate excess, but the neurosurgeon must regulate the fluid balance with much greater accuracy in order to prevent cerebral edema. After trauma to the brain, particularly where there is an accompanying reduction in the plasma protein, replacement just short of the normal hydration level is the ideal.

SUMMARY

(1) The average neurosurgical patient during the course of an extensive craniotomy loses from 200 to 1,000 cc. of water by vaporization from the skin and lungs. With a superimposed fluid loss of between 500 and 2,000 cc. from hemorrhage, the surgeon is faced with the problem of postoperative dehydration. The question of survival or death after a severe operation on the brain often hinges on how effectively water balance is restored.

(2) The loss of water vaporized from the skin and lungs can be minimized by:

(a) The substitution for ether of local infiltration anesthesia, supplemented if necessary by narcosis with basal avertin or the barbituric acid derivatives.

(b) Avoiding the use of too warm coverings in a hot operating room and subsequently in the ward.

(3) Replacement of fluid lost at operation is best carried out by the administration of a constant intravenous infusion of 5 per cent glucose in normal saline during the course of all major craniotomies. Five hundred to 1,000 cc. administered in this way will replace the insensible loss of water which has taken place on the operating table. The intravenous apparatus is also available for the transfusion of citrated blood. The infusion should be continued in the ward until a total of not over 2,000 cc. of fluid has run in. In this way the interstitial fluid reserve, as well as the electrolyte balance, can be maintained at slightly reduced but safe levels.

(4) Postoperatively, fluid must be replaced much more accurately in neurosurgical than in general surgical patients. While the latter may be given a moderate excess of fluid with impunity, a slightly deficient state of hydration is safer in patients after operations on the brain, in order to minimize cerebral edema.

(5) In those patients who are able to take fluids by mouth after operation, the problem of restoring the upset water balance is relatively simple. But the problem becomes increasingly complex in the sick or comatose patient, where a part or all of the fluid intake must be given by the intravenous route.

(6) After neurosurgical operations, a slight reduction in the concentration of sodium and chloride ions may occur, even in the absence of any upset in gastro-intestinal function. This reduction in concentration of sodium may reach the level seen in the early stages of Addison's disease, but evidence that it is harmful per se is lacking. For this reason it does not seem essential to correct the deficit unless it is aggravated by gastro-intestinal disturbances. In the absence of vomiting or diarrhea, 5 per cent glucose in distilled water appears to be a better solution for prolonged intravenous administration than when combined with normal saline, because the addition of saline increases the risk of edema.

(7) To maintain a postoperative patient in a slightly dehydrated state with safety, the surgeon must have a working knowledge of water exchange. It must be appreciated that in addition to the measured output of urine, feces, and vomitus, one to two and one-half liters are lost by vaporization from the skin and lungs in the course of each day. If this insensible loss is not taken into consideration, serious dehydration may develop.

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SURGICAL KNOTS*

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DURING the past few years much interest has been shown in regard to present day sutures. This has largely taken the form of studies on catgut and other absorbable materials. Meleney[7] as well as Clock[2] have continued their important work on the sterility of catgut and have pointed out the hazards of some of the material now on the market. Kraissl[5] has made an interesting study on some of the factors which alter the absorption of catgut. Flaws from splicing, foreign bodies and sharp kinks were found to decrease greatly the strength of a given piece of catgut. The boilable (anhydrous) type apparently absorbed more rapidly in 2 per cent trypsin than did the non-boilable type. Kraissl also demonstrated a rapid dissolution of catgut and disruption of abdominal wounds in guinea-pigs previously sensitized to the catgut. This adds weight to the opinions of others [9, 1, 4] that sensitivity on the part of the patient to catgut is an important factor in the disruption of wounds following premature absorption of the catgut.

Other protein suture materials have been suggested and used to some extent abroad. These include sutures made of milk casein, muscle tissue, nerve tissue, *etc.* It would seem that the undesirable features of a protein foreign body are thus still retained with these, giving no particular advantage over catgut.

Jenkins[4] has recently shown that 20 or 40 day catgut maintains its strength but a fraction of this time in the human body. It was further shown that plain catgut maintained its strength for four to six days. But, when the strength of the catgut depended upon a surgical knot, its strength lasted only two to three days. It failed at this early date usually because the knot became untied.

Because of the foregoing, briefly outlined disadvantages of catgut suture, there has been a noticeable trend toward nonabsorbable material. Recent advocates of the latter include Whipple,[10] Babcock,[1] and Reid, Zinninger and Merrell.[8] The strength of wounds sutured with catgut and with silk was studied by Howes[3] who found a greater advantage in the silk.

In these discussions on suture failure, one phase is noticeably absent—the least reliable part of any suture—*the knot.*

Before starting the present study it was the writer's opinion that the occasional untied knot, seen in experimental work or at the autopsy table, was the result of carelessness. It was thought that all carefully tied square or surgeon's knots stayed tied. However, at the conclusion of this study

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the startling feature seems to be that so many knots stay secure, that there are actually so few failures of abdominal midline, or rectus incisions. It is here that the suture knot receives one of its most severe tests.

Following the failure of some of our own knots, an attempt was made to review the literature on the subject. This was disappointing in that no thorough experimental work could be found. The available literature consists, for the most part, of opinions of various writers with no attempt at analysis of the physical factors involved in the knot itself. Because of an unsatisfied curiosity the following study was started.

It should be stated that this inquiry deals with suture knots which are subjected to constant or intermittent tension. It by no means considers as a serious menace those placed about the usual subcutaneous bleeders. Here, and in similar sites, any knot which will hold a few hours seems to suffice.

Methods.—Animals were first used so that the various knots could be placed under tension in conditions which closely simulated those of the human body. It soon became apparent, however, that the conditions in the animal were far too variable. The chief difficulty arose in trying to place sutures in identical mechanical, as well as physiologic, situations so as to obtain reliable comparisons. The use of animals was abandoned because of these uncontrollable variables. Their use was a test of the operator's ability to reproduce identical situations for a large number of sutures and not a true test of the knot itself.

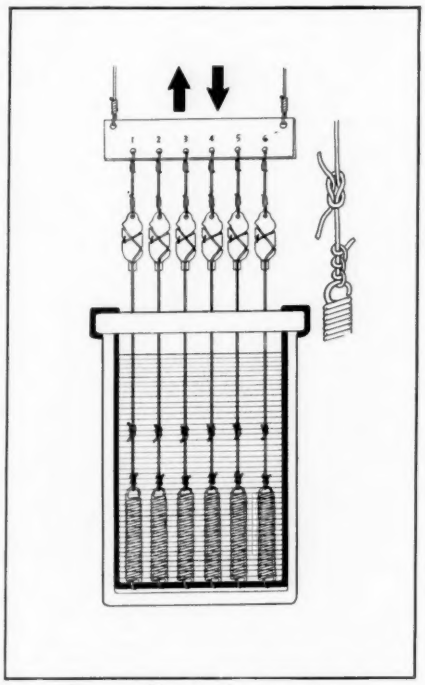

FIG. 1.—Knot testing apparatus.

The mechanical device shown in Fig. 1 was finally decided upon for use in testing the various knots. Admittedly, it leaves much to be desired, but nevertheless it gives uniform working conditions. The apparatus consists of bronze wire springs fixed at the lower end and attached to the suture to be tested at the upper end. The two ends of the suture were tied in a knot a short distance above the spring. The ends of the knot were cut to a uniform 5 Mm. length and the remaining long end brought up and laced about a fastener. This in turn was attached to the cross bar above, which was moved up and down by an electric motor at the rate of 40 times a minute. The springs were so adjusted that the upward movement of the cross bar (three-quarters of an inch) produced a two and one-half pound tension on

each suture. With the downward motion of the cross bar the suture was completely relaxed.

It seemed particularly desirable to test the knots wet as well as dry. For this purpose the springs, knots and lower portions of the sutures were immersed in a rectangular glass jar containing the desired solution. This could be done without altering the original set-up in any way. The solutions used in this phase of the work consisted of water, saline, and 2 per cent trypsin solution.

A soft rubber drag was occasionally placed in the jar so as to rub gently against the knots. This was done to simulate the rubbing of tissue against knots placed in the body. The only effect of this was to cause an early fraying of the suture adjacent to the knot with no tendency to untie it.

Knots.—The knots used were those of common surgical practice. They included the granny, square, surgeon's, and triple throw knots (Figs. 2, 3 and 4). A few modifications of these were tried but proved to be of no particular advantage. All knots were tied carefully and pulled down hard and firm.

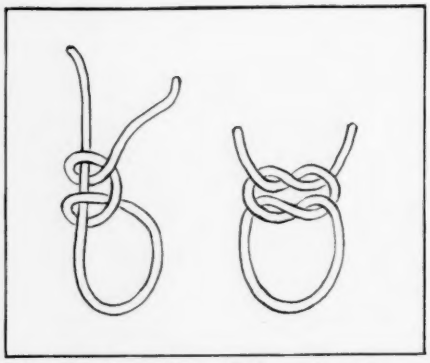

FIG. 2.—Granny knots. Both utterly useless.

The sutures consisted of 32 types and sizes of absorbable and non-absorbable material made by 12 different manufacturers. No difference was found in comparing the material made by one manufacturer with that of another provided the type of suture was the same. However, a great deal of difference was found in the different types of material and whether they were wet or dry. A detail that may be frequently neglected by the makers.

Results.—The test to which all knots were subjected is admittedly a severe one. A pull of two and one-half pounds 40 times a minute is not to be duplicated in actual practice. However, it was our experience that if the knot pulled out, it practically always pulled out during the first few pulls of the apparatus. If it held through these it would hold for hours and finally break in the portion of the suture immediately adjacent to the knot. It is evident then that if a suture in the body is subjected to a tension of two and one-half pounds on several occasions, the ability of the knot to hold can be compared directly with the results of the mechanical testing apparatus.

(1) SQUARE KNOT.—(A) *Plain Catgut:* When this material was tested dry the knot held in approximately 85 per cent of the trials, slipped a few millimeters and held in 10 per cent and pulled out in 5 per cent. The square knots which were tested dry and found to hold were then immersed in saline or 2 per cent trypsin solution. The machine was again started after a variable period of time (1 to 24 hours) and immediately 80 per cent of

these square knots pulled out. This high percentage of failures is partially accounted for by numerous trials using the nonboilable, very pliable and soft catgut. We were unable to tie a square knot in this material which would hold if the gut had first been soaked in animal serum or 2 per cent trypsin. A reliable knot could be tied dry but not wet. The slippery loops of the knot merely rolled over and over to the cut suture ends.

(B) *Chromic Catgut:* Square knots tied with this material held much better than with the plain gut. They held dry in every instance except occasionally in the larger sized sutures which were wiry and could not be pulled down tight. When knots known to be solid dry were soaked in saline or 2 per cent trypsin, approximately 25 per cent pulled out when the machine was again started.

(C) *Silk and Linen:* Knots tied from these materials were the safest

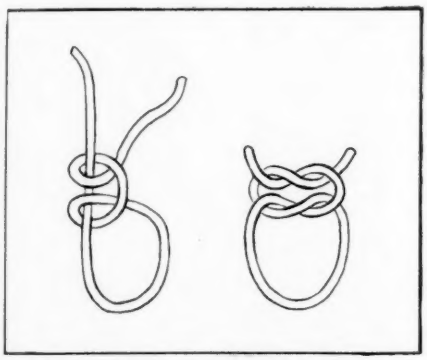

FIG. 3.—Both are square knots though the one on the left is quite useless. It is produced very frequently by those tying with one hand and keeping slight tension on the suture to keep it taut. (Right) Square knot fairly safe with most materials but not safe with the soft pliable type of plain catgut.

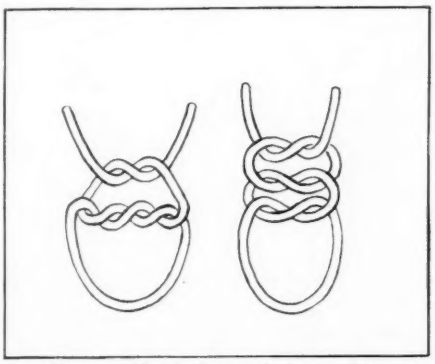

FIG. 4.—(Left) Surgeon's knot. Less reliable than square knot for catgut ties. Unless the second throw causes the first to overlap upon itself, and thus set the knot, it is very apt to loosen and give way. (Right) Triple throw knot. Safest of all simple knots although it pulls out at times when using wet plain gut and is not safe with the pliable nonboilable type.

of any tested. They were nearly 100 per cent reliable provided the cut knot ends were left 3 to 5 Mm. long.

(2) SURGEON'S KNOT.—(A) *Plain Catgut:* When tied dry, this knot was very disappointing. Its failure was considerably higher than that of the square knot. Approximately 35 per cent held, 30 per cent slipped a few millimeters and then held while 35 per cent pulled out immediately on starting the apparatus. Of knots holding when tested dry and then soaked in saline or 2 per cent trypsin, 75 per cent pulled out. Here again the percentage is unusually high because of including a number of trials with the very pliable nonboilable catgut. None of these knots held.

(B) *Chromic Catgut:* The results were practically the same as with plain (boilable) catgut with many failures of the dry and wet knots.

(C) *Silk and Linen:* The knots in these materials again were superior to the catgut. There were, however, occasional failures.

Comment.—It was quite surprising to find the so-called surgeon's knot

so unreliable. An analysis showed that the extra loop of the first throw often prevented the second throw from being pulled down firmly on the first. When the second throw is pulled back and forth in tightening, it causes the first to overlap in a hard firm knot. This will hold, but very few operators take the additional time necessary for this maneuver. The knots tied in this part of the test were tied carefully and pulled down hard and tight. No attempt was made, however, to work the suture ends back and forth to obtain a harder and more compact knot.

(3) Triple Throw Knot (Fig. 4).—This knot was by far the most reliable of the three detailed here. It was tried with four variations. These were triple throw, all throws tied square, first two throws square with third a granny, first throw a granny with second two square, and lastly a triple throw knot with none square (all grannies). Of these variations the triple throw all tied square was the safest. The least reliable was the triple throw in which none of the throws was tied square. However, this knot was quite as safe as the surgeon's knot or the simple square knot.

(A) *Plain Catgut:* This held dry in every trial except in a few instances in which the pliable nonboilable variety was tested. When the knots were soaked as previously indicated and then again subjected to the intermittent pull of the apparatus, about one-half pulled out. As already stated fewer failed in the group with all throws tied square.

(B) *Chromic Catgut:* This knot and this material were the only combination of aborbable suture which produced a 100 per cent reliable knot under the conditions of the testing apparatus. This was true wet or dry.

(C) *Silk and Linen:* Again 100 per cent reliable.

In view of the large number of knots which failed under the conditions listed, a brief analysis would seem in order. In the first place a square, surgeon's, or triple throw knot which is reliable can be tied if proper time is taken with tugging on one suture end and then the other until the throws are firmly worked together. It must be admitted that this is not the manner in which most surgical ties are made. For the most part the throws are pulled together by a hard steady pull. This was the case in our test knots, care being taken that the knots were flat and were not allowed to slide on one suture as indicated in Fig. 3.

As noted, a large proportion of the tested knots which were allowed to soak in saline or trypsin solution immediately pulled out when again subjected to tension. But, when such knots were being tested dry and solution added to the jar to moisten them during their intermittent tension, no such failure took place. The knots held firmly in 95 per cent of the trials. This probably means that as the catgut material becomes softened by the solution it is more tightly pulled into a firmer knot by the constant tension. Undoubtedly this same process must take place in conditions of actual use, or there would be far more wound failures. On the other hand when the catgut knot absorbs moisture and is without tension, the swelling

of its fibers loosens the knot leaving it quite vulnerable to any subsequent tension.

The size of the suture material played a part in the stability of the knots. In heavy wiry material such as No. 2 chromic, the knot would often not stay tight but would again spring open. Once the knot was made to stay tight and compact there seemed to be little difference between the large and small sizes using the uniform tension of two and one-half pounds.

Knot ends were cut to a length of 5 to 6 Mm. for gut sutures and 3 to 4 Mm. for nonabsorbable sutures. Shorter ends than this gave knots which were not at all reliable.

Several points were quite puzzling. Why should silk and linen knots

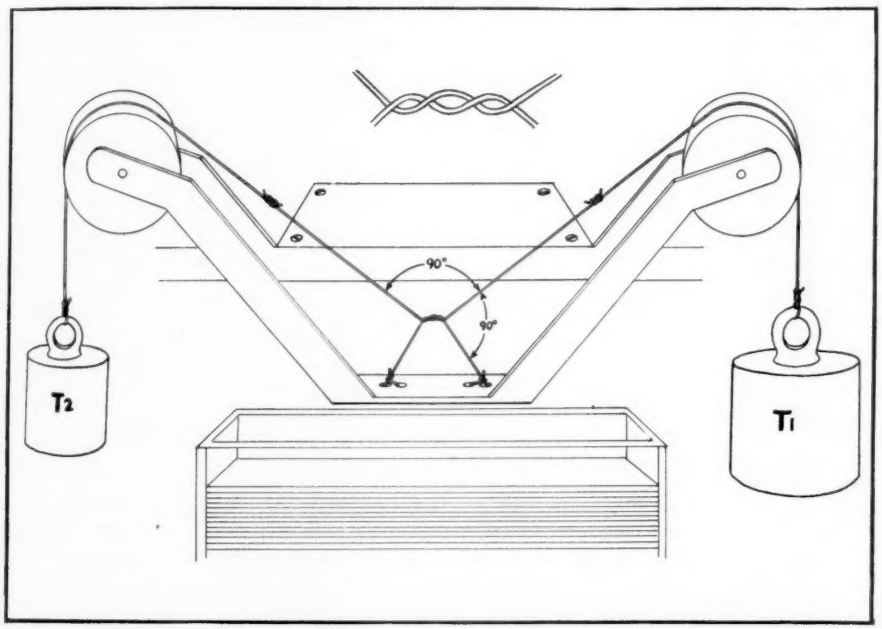

FIG. 5.—Apparatus used in determining coefficient of friction of various suture materials. When this was to be determined on wet sutures, the jar beneath was elevated so as to immerse the two strands being tested.

hold so much better than those of gut? Also why should chromic catgut give safer knots than plain? Why should the pliable nonboilable plain gut give knots which are entirely unsafe unless triple or quadruple throws are used?

Coefficient of Friction.—In the final analysis a knot in string or thread stays tied because of friction of one portion of the knot upon another. If the coefficient of friction of the particular material is great, the knot will hold firmly. If the coefficient of friction is very small, then the component parts of the knot will slide upon each other, and the knot will fail.

It was thought, therefore, that this was the deciding factor. The reason plain catgut knots failed so frequently when they were wet was that as the catgut became wet it also became slippery, and hence possessed a much

lowered coefficient of friction. While this thesis is true of nonabsorbable materials such as silk and linen, the coefficient of friction proved not to be the all important factor in catgut.

It seemed desirable then to learn the coefficient of friction of various suture materials both in the dry and wet state. Through the aid of A. A. Potter, Dean of Engineering, Purdue University, it was learned that no standard method exists for determining the coefficient of friction of silk thread against silk thread, catgut against catgut, *etc.* Furthermore, Dean Potter could learn of no available data indicating the coefficient of friction of thread against thread when placed in the form of a knot.

With no precedent to follow we felt free to design an apparatus which would best meet the demands of our particular problem. The result was the apparatus illustrated in Fig. 5. Here one strand of the suture to be tested was fastened to the base. A second strand interlaced around the first thus forming an angle of contact of 540 degrees. In most of the tests only a single loop was made (180 degrees) since the longer contact had a tendency to override and give false friction values. This was particularly true of the finer sutures. The length of the first (lower) suture was adjusted so that the two formed 90 degree angles as indicated. The free ends of the second suture were fastened to weights over freely moving ball bearing pulleys. Weight T_2 was a constant being in the various trials 50, 100, 150, and 200 Gm. Weight T_1 was then increased to a point where the second suture was about to slip through the overlapped first suture. The greatest weight which could be added without slipping was taken as T_1.

With these available figures the coefficient of friction was calculated by direct substitution in the formula used for computing belt and pulley friction[6]—

$$\frac{T_1}{T_2} = e^{fb} \quad \text{or} \quad \log_{10} \frac{T_1}{T_2} = .4343\ fb$$

In the above, T_1 and T_2 were determined experimentally; $e = 2.718$ (base of the Napierian system of logarithms); f = coefficient of friction; b = angle of suture contact in radians. All these factors are known with the exception of f (coefficient of friction). Therefore, f can be determined by substituting the values obtained for each trial run. For the sake of brevity no mathematical discussion or example computations on trial runs will be given. The average results of these trials are indicated graphically in Fig. 6.

As has been previously indicated and known to all surgeons, suture knots which are wet behave far differently than when dry. A silk suture knot apparently holds much better when wet while plain catgut which has been allowed to soak in serum is extremely hard to tie so as to be secure. It, therefore, was important to determine the coefficient of friction values for the various sutures after they had been soaked in serum as well as their values dry. Wet suture tests were made while the sutures were still immersed

in serum in a flat glass container which was elevated into place. The results are also shown for comparison in Fig. 6.

A comparison of these average wet and dry values is quite interesting. It will be noted that silkworm gut, dermal and the strong hard twist type of silk showed no increase in friction value after they had been soaked in serum. On the other hand, the coefficient of friction on linen and standard surgical silk increased approximately two and three times when wet with serum. This is rather important since it indicates that knots tied with these materials are more apt to stay tied than those with lesser frictional values. However, it also indicates another factor which tends to prevent a safe knot. When a knot is tied with linen or standard surgical silk the material is always wet or at least damp with serum. This means that it has an unusually high coefficient of friction which in itself may prevent pulling the throws tightly together without breaking the suture. Such an experience is known to all operators and is particularly common in trying to pull down snugly the first throw of a surgeon's knot. The suture often breaks, and the operating room personnel receives a vindictive blast for having boiled the silk too long.

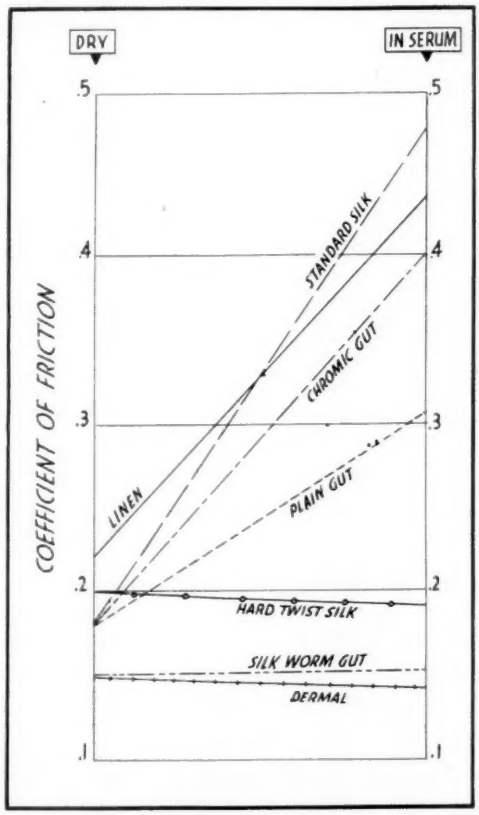

FIG. 6.—Coefficients of friction for various suture materials when in the form of a knot. On the left are shown values for the dry suture. On the right are the values for the same sutures after they have been soaked for a short time in serum. The lines between the two values merely represent graphically the degree of change.

The dry and wet coefficient of friction values for catgut were quite surprising. This was particularly true in the case of plain catgut. It was expected that this material would show a lower friction value wet than dry since grossly it seems to become more slippery and since knots pull out so much more readily wet than dry. This was not found to be the case. There was an approximate 80 per cent increase in the friction values on plain catgut as indicated in Fig. 6. The values for chromic catgut more than doubled when saturated with serum. This latter fact coincides with our knot experiments and that of clinical experience to explain the more reliable knots obtained with chromic catgut.

It should be stated that by the described method of testing for coefficient

of friction, the smaller sizes of suture material gave slightly lower values than the larger sizes. The differences were not great and have been omitted from the discussion and Fig. 6.

Suture Elasticity.—It is obvious that an adequate explanation has not been given for the more frequent failure and slipping of plain catgut suture over other materials. Especially is this true with the wet gut, for though the frictional values are greater than when dry, the simple knot is practically useless in the wet state.

There is another factor peculiar to catgut as compared with nonabsorbable materials which very definitely affects the safety of a knot. This is the resilience or elasticity of the material. It is well known that catgut swells and is considerably more elastic when wet than when dry.

It was found that a full length strand of number one plain catgut stretched from 1 to 1.5 per cent when subjected to 1 Kg. tension. Identical figures were obtained for similar chromic material. But, when these two sutures were again tested after soaking for a short time in saline, quite a change was noted. The plain gut had an average elasticity (stretch) of 15 per cent while that of the chromic was less than 7 per cent. This is contrasted with the values obtained for linen under similar conditions. These were 0.7 per cent stretch dry and 1.8 per cent wet.

The significance of the foregoing is apparent by comparison with other elastic materials. For instance, the tied ends of a rubber band readily slip out of their knot when subjected to any degree of tension. The knot does not become untied, its loops merely roll over and over each other until the knot ends are reached. So it is with catgut and particularly the pliable type of plain catgut.

One more factor seems worthy of note in regard to the slipping of catgut knots. Catgut swells and increases in size when allowed to soak in serum or saline. It was found that the diameter of boilable No. 1 plain gut increased on an average of 55 per cent. This value was somewhat lower for nonboilable and chromic catgut. It seems likely then that a knot tied with dry material may actually "swell itself loose." This is undoubtedly more of a factor with the larger sized strands.

Discussion.—Again it should be stated that this inquiry into the safety of knots and suture materials applies only to those which receive definite tension. Those used for the ligation of subcutaneous bleeders or for closing the skin do not enter into the discussion.

The high proportion of knot failures cited is in part due to the severe test to which they were subjected. It seems probable, however, that in any abdominal wound, several of the sutures and their knots may be subjected to a tension of two and one-half pounds. To these our results are directly applicable. No study was made of knot failure using tensions less than the above.

The experimental work on the cause of frequent knot failure in catgut

is principally of academic interest. It explains at least some of the causes for failure of plain catgut knots as compared with other materials.

It is believed that many of the suture failures which are seen clinically are due actually to the knot becoming untied rather than breaking or premature digestion of the catgut.

SUMMARY

(1) Many suture failures are due to the knot rather than the suture material. An inquiry is here recorded on the reliability of common surgical knots.

(2) It includes only those knots and their sutures which are subjected to an appreciable tension.

(3) The tests to which the knots were subjected are admittedly strenuous. They are, however, quite within the realm of general clinical experience.

(4) Any conclusions must be tempered by the realization that there exists a wide variation in different operators' knots and in different knots of the same operator.

(5) Notwithstanding these variations some broad generalizations may be stated.

(6) Safest of all common surgical knots is the triple throw knot (Fig. 4). This is safest when all throws are tied square but is also quite reliable if any two of the three throws are tied square.

(7) The square knot is more reliable than the surgeon's knot. The latter is difficult to pull down tight enough so that it will not slip out.

(8) The least reliable knot material is plain catgut, particularly the pliable, nonboilable type. This is seldom safe in square or surgeon's knots and occasionally fails in the triple throw knot.

(9) From the standpoint of the knot, chromic catgut is much safer than plain though only in the triple throw knot does it approach 100 per cent reliability.

(10) A marked difference is noted in the holding power of catgut knots in the dry and in the wet states. A knot which is perfectly safe when dry may readily slip out when moistened with serum.

(11) An inquiry was made into the causes of failures in the catgut knots wet with serum. Contrary to common belief, wet catgut is not more slippery than dry. In fact the frictional value increases when this material is soaked in serum (Fig. 6). It is contended that this failure is largely due to the marked increase in elasticity of the catgut (particularly plain) when wet with saline or serum. An apparatus for testing the coefficient of friction of materials tied in the form of a knot is described. Another factor in the failure of catgut knots is the swelling of the material itself when moistened (approximately 50 per cent). This, undoubtedly, has a tendency to loosen the throws of the knot.

(12) Knots made in linen or silk sutures are much safer than those of catgut. The wetting of these with serum has no tendency to cause them to

slip. Again the surgeon's knot was less reliable than the triple throw or square knot. The two last named approach 100 percent reliability when using linen or silk.

(13) Cutting the suture ends shorter than 5 Mm. for catgut and 3 Mm. for linen or silk is a distinct hazard to the knot.

(14) Knots tied in large sized material occasionally fail because the suture is too bulky or wiry to be pulled down snugly.

(15) If the knot holds, the next weakest point is immediately adjacent to it, where the suture fibers have been fractured in the tying.

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BRIEF COMMUNICATIONS AND CASE REPORTS

INTRAPERITONEAL STREPTOCOCCIC ABSCESS COMPLICATING ACUTE PHARYNGITIS*

ACUTE ILEUS. OPERATION. RECOVERY

ARTHUR S. W. TOUROFF, M.D.

NEW YORK

Case Report.—M. S., female, married, age 38, was admitted to the Mount Sinai Hospital at midnight, April 10, 1934, with a history of having developed an acute pharyngitis, characterized by sore throat and mild fever, four days previously. Symptoms continued for three days, at which time the temperature became higher and she began to suffer dull abdominal pain. This was most marked in the lower half of the abdomen and was not accompanied by nausea or vomiting. Within several hours, the pain became severe and cramp-like, and temperature rose to 105° F. The past history was essentially negative except for an induced abortion five years previously. There had been two other pregnancies, both children living and well. The venereal history was negative.

Physical Examination.—The patient appeared acutely ill. Temperature 105° F.; pulse 136; respirations 28. The pharynx and tonsils were deeply congested. A purulent, postnasal drip was present. The abdomen was slightly distended, tender, and moderately rigid throughout, the physical signs being most marked in the right lower quadrant. No masses were palpable. On pelvic examination, the cervix was tender. There was no vaginal discharge. In the right adnexal region, a slightly tender mass about four inches in diameter was palpable. On the left side, a similar but smaller mass was present.

Laboratory Data.—White blood cells 33,000; polymorphonuclear leukocytes 92 per cent, of which 66 were segmented and 26 were nonsegmented. Lymphocytes 8 per cent; red blood cells 4,850,000; hemoglobin 90 per cent; sedimentation rate 20 minutes. Blood pressure 86/60. Urine acid, specific gravity 1,022, trace of albumin, occasional white blood cells and hyalin cast. Weight 130 pounds.

The first impression was that of a patient suffering from acute, bilateral, adnexal inflammatory disease with secondary lower abdominal peritonitis, but that, in view of the preceding history of an acute pharyngitis, the diagnosis of pneumococcus or streptococcus peritonitis was to be strongly considered. In view of the positive pelvic findings, the patient was admitted to the gynecologic service.

The patient was immediately placed upon a continuous intravenous infusion of 5 per cent glucose, and the routine treatment of pelvic peritonitis. During the first week the temperature ranged between 101° and 106° F. By the end of the second week, the temperature gradually assumed a lower level and began to range between 100° and 102.6° F. In the meantime the blood culture, taken at the time of admission, was reported negative, and the throat infection had subsided completely. The diffuse abdominal signs gradually receded, but tenderness and slight spasm still remained in the right lower quadrant. Meanwhile, a firm pelvic exudate developed behind the cervix. Over a period of five weeks, the temperature continued between 100° and 102.6° F. By this time the pelvic exudate increased in size and now extended completely across the pelvis from one

* Presented before the New York Surgical Society, October 13, 1938. Submitted for publication December 29, 1937.

side to the other. There was, however, not sufficient evidence of softening at any time to warrant surgical drainage. Because of a gradually falling hemoglobin, which finally reached 60 per cent, a transfusion of 500 cc. of citrated blood was given. At the end of the seventh week of illness, the temperature again became septic in type and began to range between 99° and 104° F. At that time, increasing abdominal distention appeared and the patient began to vomit. This at first was only occasional, but it became more frequent and finally necessitated the use of an indwelling Levin tube and the administration of fluids intravenously. Abdominal distention became marked, and enemata now were only partially effectual. Examination of the abdomen revealed the presence of an intra-abdominal fluid wave which, during the course of a week, became marked. At that time (eight weeks after admission) surgical consultation was requested. When seen by the surgical service the patient presented an unusual clinical picture. She was markedly emaciated and had a large, tense, protuberant, diffusely tender abdomen, within which a fluid wave could be readily elicited. The indwelling Levin tube was draining large amounts of upper intestinal secretion. On pelvic examination, no definite pelvic exudate was palpable, there being only an indefinite sense of resistance and tenseness throughout. Temperature was 104° F.; pulse 124; respirations 36. From the history and physical examination it appeared that the patient was suffering from a diffuse, low grade, purulent peritonitis associated with a large amount of intraperitoneal pus and a complicating ileus. Abdominal puncture was performed and a syringeful of thick, greenish-yellow, odorless pus was aspirated, a smear of which showed gram-positive cocci in chains, which were later identified as hemolytic streptococci.

Operation.—June 13, 1934: Under local anesthesia, a one and one-half inch McBurney incision was made and the peritoneum exposed. Aspiration disclosed pus at once. The opening was enlarged, and 5,100 cc. of pus evacuated. The previously protuberant abdomen became scaphoid. A long curved instrument, passed into the wound, extended across the abdomen into the left lower quadrant and well upward toward the diaphragm. The finger appreciated a large smooth-walled cavity, the limits of which could not be determined. No intestines were visible, and it was apparent that the latter had been pushed posteriorly, and lay behind a pyogenic membrane which formed the posterior limit of the cavity. The condition was, therefore, a huge intraperitoneal abscess lying in the anterior portion of the abdomen. Because of the patient's poor general condition more complete exploration was not attempted. No counterdrainage was introduced, the thought being to tide the patient over the critical period and to establish more thorough drainage at a later date. An open end, large caliber, mushroom catheter was introduced into the abscess cavity and the superficial wound packed with iodoform gauze.

There was practically no postoperative reaction, and within three days the temperature fell below 100° F. The symptoms of ileus subsided almost at once. Purulent drainage through the tube diminished from 20 ounces on the first postoperative day to two ounces on the fifth day. Eight days after operation, roentgenologic examination of the abdomen was made after the injection of lipiodol into the cavity through the wound, in order, if possible, to determine the limits of the cavity and the best sites to introduce counter-drains. The films revealed the lipiodol extending irregularly from the right to the left limits of the abdomen and from the pelvis upward to the region of the diaphragm. Because of its large size no attempt was made to fill the cavity completely.

Second Operation.—On June 26, 1934, 13 days after the original operation, under avertin-gas-oxygen anesthesia, three counterincisions, one in the right lower quadrant, another in the left lower quadrant, and a third in the left upper quadrant, were made. At this time the interior of the cavity was explored more completely, and the limits found to correspond with those demonstrated by previous roentgenologic examination. The intra-abdominal viscera were all found displaced posteriorly and could not be identified owing to their being covered by a pyogenic membrane which formed the posterior wall of the cavity. Approximately at the level of the umbilicus, there was a small defect in the membrane, through which several matted knuckles of intestine were visible. The wounds were packed with gauze.

Following operation the patient reacted slowly, apparently due to an overdose of avertin, although the amount given was presumably within safe limits. Within 24 hours she had recovered completely from this episode, but then developed a toxic-exhaustion psychosis, which continued for about a week, but responded promptly to a transfusion. From that time on, her course was entirely satisfactory. About two months later one of the small knuckles of bowel, which was noted at operation, perforated, apparently as the result of pressure of a small tube drain. A small amount of fecal discharge continued for one week when the fistula closed spontaneously.

The patient was discharged from the hospital on the eighty-fourth day following the second operation, at which time, two small granulating wounds and a residual cavity, with a capacity of two ounces were present. Her weight was 104 pounds, a gain of 29 pounds from her recorded low of 75. After leaving the hospital, her wounds were dressed in the Out-Patient Department, from which she was discharged completely well after six weeks. She has been followed in the Return Clinic for three and one-half years, to date, during which she has remained entirely asymptomatic. Her present weight is 135 pounds.

DISCUSSION.—The above case is of interest for several reasons: First, it illustrates an unusual cause of acute ileus. At the time of operation, ileus, while not complete, appeared to be rapidly becoming so. Whether the ileus was of the so-called dynamic or adynamic type cannot be stated, but from the nature of the intraperitoneal lesion it may well have been either. Secondly, the case is of interest because it illustrates an unusual lesion. While streptococcic peritonitis following upper respiratory infection is not uncommon in children, it is rare in adults. Super-added, there was the development of an unusual lesion: namely, a massive intraperitoneal abscess which filled the entire anterior portion of the abdomen. Finally, the case illustrates the value of diagnostic abdominal aspiration as described by Drs. Harold Neuhof and Ira Cohen some years ago. If this procedure had been performed at the time of the patient's admission to the hospital, the diagnosis of streptococcic peritonitis would have been made immediately.

DISCUSSION: DR. HAROLD NEUHOF (New York) referred to a roentgenogram of the chest of the case cited which presented a very interesting feature, which might possibly throw some light on the process of the evolution of this interesting abdominal lesion. The patient's condition began with an upper respiratory infection (acute pharyngitis). During her hospitalization a pneumonic process in the right lower lobe with some pleural exudate was demonstrated. Subsequently this bronchopleural process subsided, but it seems that in this, as in a few other similar cases which he could recall, there is a possibility that the pathway of infection was from the mediastinum into the posterior retroperitoneal spaces. Thus the intraperitoneal exudate would be a by-product of the retroperitoneal pathologic progress. Doctor Touroff exercised sound judgment in not exploring widely in order to determine just to what extent the peritoneal cavity was invaded.

With regard to the possibility of diagnosing cases of this kind at a much earlier stage of the intraperitoneal process than was done in the present instance, Doctor Neuhof advocated the use of abdominal puncture for the diagnosis of certain cases of acute intraperitoneal infection. If the case cited had had an abdominal puncture performed at an early stage, it is possible that the process would have been recognized and the patient subjected to operation considerably earlier than she was. In many cases the nature of the infection

can be definitely established in this way, and certainly the question of whether or not to proceed with operation can often be determined.

Dr. Sigmund Mage (New York) cited the case of a white woman, age 31, who was originally seen five weeks after the onset of her illness. She had been first treated at the Morrisania Hospital where she had been admitted five days after the onset of an upper respiratory infection associated with upper abdominal cramps and diarrhea. The impression then was that the patient was suffering from pneumonia associated with general peritonitis. On this basis, she was treated in an oxygen tent for three days, following which her respiratory affection apparently cleared up. The abdominal distress, cramps and signs of abdominal fluid, however, persisted. She was transferred to Doctor Neuhof's service at Mt. Sinai Hospital approximately four weeks after her admission to Morrisania Hospital. She then presented a picture of an acutely ill patient. Temperature was 102° F., white blood cells 26,000, 80 per cent polys. The abdomen was markedly distended, and there were present signs of fluid. The impression was of a patient suffering from a tuberculous peritonitis. An abdominal puncture was performed and a thick, creamy pus was obtained, which on smear revealed extracellular gram-negative diplococci. Under local anesthesia, a small McBurney incision released six liters of thick, creamy pus. Doctor Mage's impression was that he was dealing with a large abscess cavity with the gut compacted posteriorly into as small a space as possible. A small counterincision was made on the left side. A tube introduced passed through both incisions; 200 cc. drained during the first 48 hours. Recovery was uneventful and the patient was discharged six weeks after admission. When seen six months ago she was in excellent health. The culture proved to be a pneumococcus Type I.

RUPTURE OF INTESTINE INTO ABDOMINAL SCAR *

Sigmund Mage, M.D.
New York

Case Report.—The patient, a white male, age 35, was first admitted to the Beekman Street Hospital May 7, 1936, with a six hour history of severe right upper abdominal colic associated with several vomiting attacks. The findings were those of an acute cholecystitis associated with an acute respiratory infection. The symptoms progressed rapidly so that within 24 hours exploration was deemed imperative, and a cholecystectomy with drainage was performed for a completely gangrenous gallbladder. The patient's immediate postoperative course was relatively smooth except for a persistent cough.

On the ninth postoperative day some separation of the skin was noted at the upper end of the wound just below the site of the drainage tract.

On the twelfth postoperative day the skin separation had extended for at least one-third of the extent of the incision, and a granulating area was seen in its depths which suggested intestine. The condition was considered a partial dehiscence without a prolapse of intestine or compromise of its function.

By June 5, 1936—28 days after operation—the patient was discharged with the wound healed except for a superficial separation of the skin edges. He attended the Out-patient Department twice a week for a month, at the end of which time complete healing had taken place, and he was able to return to work.

On August 8, 1936—74 days after his discharge from the hospital—he was readmitted,

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stating that: While helping in the lifting of a heavy ladder he suddenly became aware of a moistness on his abdomen and discovered a profuse drainage coming from an opening in the abdominal scar. While there was a consciousness of a straining effort, there was no particular pain accompanying the experience. He returned immediately to the hospital where examination revealed a thin biliary-like discharge containing some food particles coming from the old drainage tract. Methylene blue given by mouth appeared in 45 minutes through this site and established the presence of an intestinal communication. Despite the immediate application of a protective coating of bronze paste, the drainage caused a progressive digestion of the skin which, within a few days, involved most of the upper abdominal wall.

This was finally controlled by introducing a continuous drip of one-tenth normal hydrochloric acid into the fistulous opening and plugging it by gauze soaked in bovinine. This was started one week after the patient's admission, and the healing of the skin progressed rapidly except for an area of painful irritation immediately about the fistulous opening. The patient had a striking mental reaction to this discomfort, becoming uncooperative and threatening suicide unless relieved. This prevented any further delay for better skin healing and, on September 15, 1936—27 days after his second admission—he was operated upon. There was an obvious ventral hernia, but because of the condition of the abdominal wall, it was felt wise to limit the procedure to the intestinal problem.

Operation.—A nine inch segment of small intestine—apparently lower ileum—with a 2½x5 cm. defect in its antimesenteric aspect was found to be firmly fixed in a hiatus in the abdominal wall musculature. Upon releasing this loop, it was evident that the intestinal rupture could not be closed without compromising its lumen. A segment six inches long involving the defect was, therefore, resected and continuity of the intestinal tract reestablished by a side-to-side anastomosis. The abdominal wall was closed by through-and-through heavy silk sutures without attempting to correct the ventral weakness.

The patient's postoperative course was uneventful except for some separation of the wound which healed without any complication. He was discharged October 13, 1936—28 days after operation. He has remained well but has a large ventral hernia which is to be repaired at some future date.

This case is of interest because an intestinal rupture through an abdominal scar is apparently a very unusual complication. A cursory search of the English literature for the past ten years has revealed no similar cases. The mechanism, of course, is apparent: namely, a muscular strain tearing the wall of a fixed, prolapsed loop of intestine which had most probably been weakened by previous infection. It is surprising, with the high incidence of recognized and unrecognized wound dehiscences and the large number of postoperative herniae, that this type of complication is not seen more often.

SPONTANEOUS RUPTURE OF THE LIVER IN MALARIA

CASE REPORT

JOHN S. MCEWAN, M.D., AND DUNCAN MCEWAN, M.D.
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RUPTURE of the liver often presents itself to us in traumatic cases and occasionally we may see or read of a case of spontaneous rupture of the liver. The spontaneous rupture is usually the result of syphilis, either hereditary

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or acquired; or of carcinoma, either primary or metastatic. It has rarely been reported in other conditions.

E. Devic and L. Beriel,[1] in 1906, reviewed the literature and collected several cases of spontaneous rupture. The apparent causes of these varied: One occurred from obstruction due to gallstones; one was reported in a patient with pleurisy and pericarditis, where the liver was said to have been very friable as the result of high temperature; another occurred on the twenty-ninth day of convalescence from typhoid fever; others were due to either carcinoma or syphilis. Nauwerck[2] reported a case due to bile stasis from stone obstruction; Walz,[3] in 1921, records a case due to multiple aneurysms of the liver; and Mast,[4] in 1933, had a case resulting from primary carcinoma of the liver.

Maze[5] gives perhaps the most logical explanation of the pathology of this condition: "In traumatic rupture, the rupture is the cause of the hemorrhage. In spontaneous rupture, the hemorrhage is the cause of the rupture. The chain of events leading to rupture are infarct, hypervascularization at the periphery, rupture of a vessel, intrahepatic hemorrhage, with resulting rupture of the tissues and production of a subcapsular hematoma, which, when rupturing the capsule permits escape of blood into the peritoneum leading to peritonitis and death."

We have been unable to find any reports of spontaneous rupture of the liver resulting from malaria.

Case Report.—H. B., female, age eight, was admitted to the Orange General Hospital at 10 A.M., March 3, 1931, complaining of severe abdominal pain. Two weeks before admission, she had been seized with a sudden sharp pain in the left upper abdominal quadrant, which radiated to the right lower quadrant. This was accompanied by a slight fever, but there were no other accompanying symptoms. The following day the pain had decreased in intensity, but was still present in the left upper quadrant. A diagnosis of tertian malaria was made, after a positive blood smear.

Following the administration of quinine the patient developed pain in the right upper quadrant, just beneath the costal margin in the gallbladder region. The pain was constant, but was worse on alternate days, becoming progressively more severe, and was accompanied every other day by a temperature of 100° to 101° F. She had no chills at any time. Two weeks after the onset of her illness, March 2, 1931, the patient experienced a very acute pain on the right side of the abdomen, broke out in a cold sweat, and had to sit up rather than lie down. That evening the pain became less, but she could not sleep. The following morning, however, the pain had become more severe and she was brought to the hospital.

Her past history was essentially negative except that she had always been thin and frail. Since a tonsillectomy six months previously, however, she had been gaining weight and feeling better. There was no history of trauma or injury. He family history was negative and the parents showed a negative blood Kahn reaction.

Physical Examination showed a patient, apparently in a critical condition, who appeared pale, restless and anxious. Her examination was essentially negative except for the local abdominal condition. The abdomen was distended and slightly rigid, the rigidity being more marked over the right side. There was tenderness over McBurney's point and also just beneath the right costal margin, but rigidity precluded the palpation of any masses. The spleen was markedly enlarged and tender. The temperature on admission was 103° F., pulse 136, respirations 40. Leukocyte count 13,800, with 85 per cent poly-

nuclear, 9 per cent small and 6 per cent large lymphocytes. A smear did not show any malarial parasites at this time. The urine was acid, specific gravity 1,019, clear, one plus albumen and an occasional pus cell. A tentative diagnosis of a ruptured appendix was made.

Operation.—Under nitrous oxide and oxygen anesthesia plus local novocain infiltration, the abdomen was opened through a right rectus incision, and a quantity of dark red blood was encountered. The appendix was normal. On extending the incision upward, a clot was exposed extruding from a rupture of the right lobe of the liver, near the inferior margin and the anterior surface. The liver was greatly enlarged, as was the spleen. The clot was removed and a rent in the liver approximately 2x2½x½ inches was revealed. The tissue seemed broken down. This cavity was cleaned out and packed with gauze and the abdomen closed. The patient left the table in very poor condition and in spite of supportive measures, expired 15 hours later. Permission for necropsy was not obtained.

COMMENT AND SUMMARY.—The case is of interest because there have been no instances reported of spontaneous rupture of the liver in malaria. The history of intermittent fever, the finding of tertian malaria plasmodia in the blood, and the enlarged spleen and liver present the malaria picture. Undoubtedly focal necrosis took place in the liver substance, resulting in hemorrhage, distention of the capsule and subsequent rupture. This but adds another complication of malaria, and another cause for an "acute abdomen."

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INJURY OF THE LIVER

REPORT OF TWO CASES

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FROM THE SURGICAL CLINIC OF THE PETER BENT BRIGHAM HOSPITAL, BOSTON, MASS.

HEPATIC trauma is of rather common occurrence at the present time, as shown by the recent survey by Krieg.[1] However, it seemed of interest to report the two following cases because of certain unusual findings. The mortality in unoperated cases probably reaches 80 per cent, and in those subjected to operation approximately 60 per cent.[2] For this reason one must

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continuously bear in mind the possibility of such an injury in cases appearing in emergency wards, following accidents.

Case 1.—J. V. S., male, age seven, was admitted to the Peter Bent Brigham Hospital August 8, 1936, at 10 A.M. Shortly before this, the boy had been struck by an auto, the front and rear wheels of one side passing over his body.

Physical Examination.—The patient was conscious but pale and very restless. His extremities were cold and clammy. Blood pressure 60 Mm. of Hg. systolic; pulse 130. The abdomen showed diffuse tenderness and moderate spasticity in both upper quadrants.

The patient was placed in moderate Trendelenburg position, warm blankets were applied, and he was given 1,000 cc. of 5 per cent dextrose intravenously. His condition improved somewhat. At the end of two hours, in spite of this treatment, his pulse increased, while his blood pressure continued to remain low. He was, accordingly, given 200 cc. of blood. His condition remained unchanged until about 6 P.M., at which time there was again an increase in the pulse rate. The patient complained of pain in the epigastrium, and at this time a definite increase in the size of the liver could be made out. A diagnosis was made of rupture of the liver, and immediate operation advised.

Operation.—Under ether anesthesia, the abdomen was opened through a midline incision. The peritoneal cavity contained a large quantity of blood. Exploration revealed that the entire left lobe and a portion of the right lobe of the liver had become nearly completely separated. This fracture ran from the point of entry of the round ligament of the liver, posteriorly and to the right, so that approximately one-half the liver substance was lying free, with the exception of a small point on the posterior, upper aspect. At this time the patient's blood pressure suddenly fell, bleeding was controlled by pressure with a sponge on the raw surface of the liver and he was then given 500 cc. of blood. It was possible after this to carry out the rest of the operation without undue incident. The small bridge of liver tissue which remained was doubly clamped and cut; the raw area of the liver was then closed by means of mattress sutures. A narrow gauze drain was introduced and the abdomen was closed in layers.

Pathologic Examination.— Gross.—The weight of the specimen removed was 354 Gm., and measured 17x9x6 cm. The liver in a boy of this age according to Coppeletta and Wolbach[3] is 680 Gm., so that in this case approximately one-half the liver substance was lost.

On August 9, the day following operation, the patient's condition was good. However, his pulse rate was still rather high, and for this reason he was given another small transfusion of whole blood. The drain was removed on the seventh postoperative day, and following this there was bile-stained drainage for nearly two weeks. The results of the urine and blood chemistry studies were normal throughout his hospital stay. Six months after the injury his condition was excellent.

Case 2.—M. L. G., female, age 19, was admitted to Lakeside Hospital September 21, 1931, at 6 P.M. About 5.30 P.M., while driving, her car ran off the road, turned over and pinned her beneath it. She was able to stand up, and was immediately hospitalized. When first seen she seemed in moderate shock; the blood pressure was 90/60; and she seemed cold. She was placed in bed and kept warm.

Physical Examination showed crepitation at the junction of the cartilage and eighth rib, tenderness over both lower rib margins and board-like abdomen. There were also multiple bruises and lacerations. At the end of five hours the blood pressure had returned to a normal level and the pulse was 100. However, the white blood count had risen to 19,000, and the red blood count was 3,700,000, hemoglobin 65 per cent.

On the following morning her condition was much improved. Respiration, pulse, blood pressure and temperature were normal. The abdomen was still rigid. The patient's condition did not improve during the day and by evening the temperature had risen to 102° F. The leukocyte count remained at 19,000, and the red cell count at

3,500,000. There was definite discomfort in the right and left sides. Exploratory operation was carried out with the preoperative diagnosis of a ruptured viscus.

Operation.—Under gas-oxygen-ether anesthesia, the abdomen was opened through a right midrectus incision and was found to be filled with old blood. The liver on the right side was normal. The blood seemed to be coming from the left upper quadrant, which was found to be filled with bile-stained blood and bile. A fragment of necrotic, yellowish liver, as large as the palm of the hand, was lying free on the spleen. Another fragment of necrotic liver was still attached to the left lobe of the liver, from which it was almost entirely separated by a large fissure. The loose fragment and all possible blood were removed from the abdomen. No bleeding points were seen. The incision was then closed in layers, without drainage.

The postoperative course was uneventful and temperature gradually subsided. The wound healed per primum. The patient was discharged on the fifteenth postoperative day.

The first case is of interest because the patient lost approximately one-half of his liver, and in spite of this an uneventful recovery ensued. The fact that the urine showed no gross abnormality indicates a considerable portion of liver must be destroyed before the so-called "liver-kidney" syndrome will appear.

In the second case the patient made an uneventful recovery in spite of a piece of necrotic liver which was not removed at the time of the operation. There was nothing following operation that would suggest bile peritonitis.

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MEMOIR

DUNCAN EVE

1853-1937

EIGHT years after retirement from a very active surgical practice and the onerous duties of a teacher of surgery for many years, Duncan Eve died after having completed a distinguished career, February 15, 1937.

DUNCAN EVE, M.D.

He was born at Augusta, Georgia, where his father, famed Paul F. Eve, medical hero of six wars, has a monument erected in his memory by Poland for his services as a surgeon in that unhappy country's resistance to Russia. He was graduated from the University of Nashville in 1872, where, prior to

the war, his father as Professor of Surgery had taught the largest medical class ever assembled in the South, numbering 454. He was also graduated from Bellevue Medical College in 1874. Shortly after, this ambitious son began his career as a teacher. Those were the days when the proprietary medical school was the only source of medical instruction and filled a very important need during that era. Medical science had not developed to such a highly technical and comprehensive degree. It was the glorification of the preceptorship and taught the practice of medicine and surgery as it was then known. The personality of the teachers, together with their experience and judgment, formed the basis of medical teaching. Those men were forceful, dynamic, and towering in their dominance of catastrophes. Diseases, which they did not wholly comprehend, were treated and often mastered by sheer personality and common sense. Doctor Eve, early, had the realization that medicine should be under the aegis of the university.

The younger Eve organized the medical department of the University of Tennessee in Nashville in 1880. For many years he was the Dean of the department of surgery. His work was prodigious and his fame as a teacher and surgeon was far-flung.

In later life he became professor of surgery in Vanderbilt University and still later was instrumental in combining the faculties of the three medical schools then existing in Nashville into Vanderbilt University, at which time the University of Nashville went out of existence and the University of Tennessee Medical Department was transferred to Memphis. In this movement, and all others pertaining to the advancement of medicine, Doctor Eve was a great champion. He was a gifted organizer and a shining example of the older methods of medical teaching.

Doctor Eve lived through the evolution of surgery of the emergencies into the surgery of pathologic conditions with the formal surgical attack based upon the then recent introduction of asepsis and the beginning of modern surgery. He was a great figure in the surgical development of the South. His colleagues freighted him with many splendid honors. He was made president of the state, sectional, and national societies. As Vice-President of the American Medical Association, he presided after the death of the president.

For many years Doctor Eve was chief surgeon of all the railways centering in Nashville. He was instrumental in organizing the first society of railroad surgeons, in Tennessee as well as the Southern States Railway Surgeons. As a result of his labors, transportation of the wounded and injured was perfected and their first aid and physical care forwarded. A superior authority on fractures, he also was a link between the surgery of the blood vessels and aneurysm and the newer developments in the surgical cure of diseases of the blood vessels. He contributed the chapter on aneurysms in Park's "System of Surgery." His name and work have been carried on by Dr. Duncan Eve, Jr., his only son.

Great hearted, courageous, tireless, and enthusiastic in the practice of surgery and in its teaching, he has left an indelible memory and imbued with high ambition the large classes which he taught from the south and southwest. His name was a household one in medicine and one of great distinction in the community where he lived and wrought.

WILLIAM D. HAGGARD

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